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ON

SCLEROSIS OF THE SPINAL CORD:

INCLUDING

LOCOMOTOR ATAXY, SPASTIC SPINAL PARALYSIS, AND
OTHER SYSTEM-DISEASES OF THE SPINAL CORD:
THEIR PATHOLOGY, SYMPTOMS, DIAGNOSIS,
AND TREATMENT.

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BY

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PREFACE.

THE subject of the present volume is one to which I have devoted much attention, and which I have had large opportunities of studying during the last five-and-twenty years; and I hope that my labours may go some way towards dispersing the obscurity which at present hangs over this department of medical science. I shall be especially gratified if my description of the protean forms which Tabes Spinalis or Locomotor Ataxy is found to assume in practice will in future lead to a readier recognition and a more successful treatment of that terrible malady. No one can be more fully aware than I am of the gaps which are still left in our knowledge of several of the spinal affections treated of in this volume; and I have been particularly careful to indicate these in the course of my disquisitions, in the hope of giving an incentive to further work in a direction where it is most needed.

48, HARLEY STREET, CAVENDISH SQUARE, W.
September, 1884.

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ON

SCLEROSIS OF THE SPINAL CORD.

CHAPTER I.

INTRODUCTION.

THERE is no pathological term which is now-a-days so freely and somewhat loosely used in speaking and writing about the diseases of the nervous system as the word sclerosis. We hear and read not only of primary and secondary sclerosis, but also of posterior and lateral sclerosis, of descending and ascending, and of insular, disseminated and amyotrophic sclerosis. From having been at first simply used for designating certain morbid appearances found post-mortem in various portions of the nervous system, the term has gradually come to be employed for the nomenclature of diseases, tabes dorsalis or locomotor ataxy being now frequently called posterior sclerosis, and spastic paralysis being termed lateral sclerosis. In practice, any obscure and chronic case of nervous disease is now often spoken of as "a case of sclerosis," which is supposed to explain everything, and it cannot be denied that there is a good deal of confusion in the professional mind about what sclerosis really means and comprehends; while the subject is so important that clear notions concerning it are highly desirable. Much that is new has recently been added to this department of pathology, and has, as usual, led to considerable improvement in diagnosis and treatment.

Although the term sclerosis (from σκληρὸς, hard), or induration, is therefore now applied to a great variety of diseases of the spinal cord, it should be understood that we do not invariably find a very pronounced hardening of the structures which are affected in such cases. In some few instances, indeed, and notably so where the disease is comparatively fresh, the parts may actually appear to be softened; but where the morbid process has been going on for a considerable time, as is, indeed, most frequently the case, the diseased tissues are decidedly tougher and harder than the normal ones, and may therefore be properly called sclerosed.

By sclerosis of the spinal cord I understand, then, *an irritant morbid process, standing intermediate between inflammation and simple atrophy, which invades certain well-defined and evolutionally, anatomically, and physiologically distinct areas or systems of that organ; and which leads in course of time to disintegration and wasting of the nerve-tubes, very generally to partial or complete destruction of the axis-cylinder, and to overgrowth of connective tissue.* The best known of these affections is one with which we have long been familiar as *tabes dorsalis*, more recent synonyms being *progressive locomotor ataxy* (Duchenne), *progressive locomotor asynergy* (Trousseau), *sclerosis of the posterior columns* (Erb), and *posterior leuco-myelitis* (Vulpian). The name locomotor ataxy does not appear to have been happily chosen, as the ataxy of gait, which constitutes the most prominent symptom of the fully-developed malady, is in many cases absent for years after the outbreak of the disease; the same applies to Trousseau's "asynergy"; while the other terms just mentioned only refer to the pathological anatomy of the complaint. The old term "tabes" therefore appears to be the best, and is now again gradually replacing the more recent appellations which had for some time past reigned supreme in the text-books and medical journals.

An apparently analogous affection of the lateral columns of the cord has only recently been introduced into our system of nosology by Erb, who has called it *spastic spinal paralysis*; while Charcot, who had investigated this subject about the same time independently, termed the disease

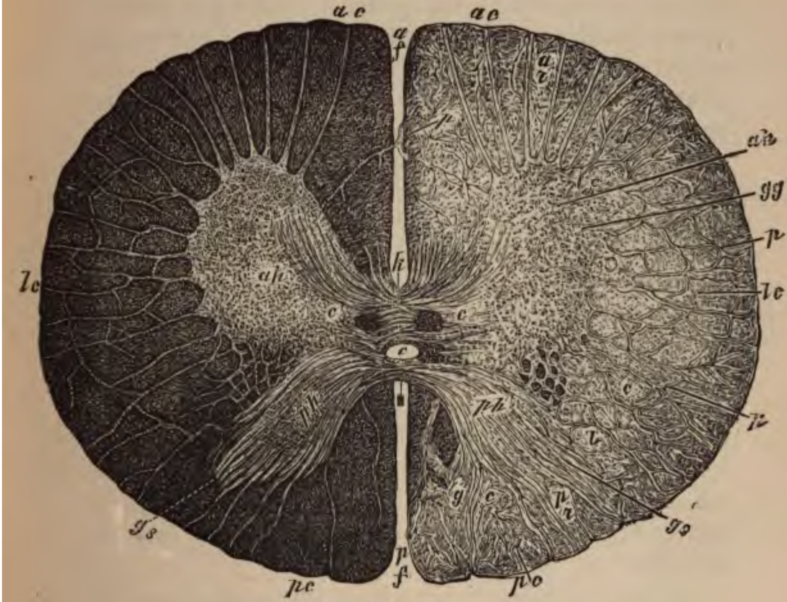

FIG. 1.

Transverse section of the spinal cord of the adult: *af*, anterior fissure; *pf*, posterior fissure; *c*, spinal canal; *ac*, anterior columns; *lc*, lateral columns; *pc*, posterior columns; *ah*, anterior horns; *ph*, posterior horns; *gs*, gelatinous substance; *cc*, Clark's columns; *gc*, Goll's columns; *bc*, Burdach's columns; *k*, anterior commissure; *gg*, ganglion cells of the anterior horns; *pp*, bloodvessels of the pia mater; *ar*, anterior roots; *pr*, posterior roots.

spasmodic tabes dorsalis. Synonymous terms are *primary lateral sclerosis* (Berger), and *sclerosis of the pyramidal strands* (Dreschfeld). Leyden, however, denies the existence of this affection as a separate disease; and as only slight evidence for it has as yet been furnished by morbid anatomy, the term "spastic paralysis" appears preferable, as it does not commit us to any definite theory about the nature of the malady.

No doubt exists about the occurrence of a combined affection of the white lateral columns and the grey anterior cornua, which was first distinguished by Charcot, and termed by him *amyotrophic lateral sclerosis*, the chief symptoms being motor paresis and rigidity, followed by atrophy of the muscular fibre. Another primary affection of this kind is *multiple*, *insular*, or *disseminated* sclerosis, which is also called *sclerosis in patches*, and which may affect not only the different portions of the cord, more especially its antero-lateral columns, but also the medulla oblongata, pons varolii, the central white substance of the cerebellum, and the medullary substance of the cerebral hemispheres. In this disease the morbid process is, as a rule, not so severe as in tabes; for while in the latter the nerve-fibre eventually perishes altogether, that important portion of it which is known as the axis-cylinder appears often to be spared in insular sclerosis. There are further anatomical differences between this and other forms of sclerosis, which will be fully described hereafter.

All forms of primary sclerosis are symmetrical, and only exceptionally confined to one half of the organ.

Secondary sclerosis, on the other hand, is mostly unilateral, and is the result of some other primary disease, such as hæmorrhage into the brain, softening of the cerebral substance from embolism or thrombosis of blood-vessels, or of hæmorrhage into the substance of the cord, and acute myelitis, Pott's disease of the vertebræ with compression of the cord, etc. When it occurs after destructive lesions of the motor area of the brain, it affects only that portion of the cord which corresponds physiologically to the diseased hemisphere, that is, the crossed pyramidal column, and Türck's anterior or the direct pyramidal column. In these latter strands the disease occurs in a descending direction, while where secondary sclerosis affects the postero-internal or Goll's columns, it takes place in an ascending direction.

Clinical observation as well as morbid anatomy seem to point to the conclusion that all these different diseases are very closely allied to each other in character and appearances, and differ more particularly according to localisation.

It is a fact, which stands out more clearly as our knowledge advances, that fibres which have certain functions in common are liable to become diseased together at exceedingly different levels of the cord, while others in their immediate neighbourhood, which have different functions, are spared. Moreover, such fibres are liable to be affected symmetrically in the two lateral halves of the organ, the degree of the morbid change being often exactly parallel in both sides of the cord. The differences in the physiological function are naturally correlated to evolutional, anatomical, and possibly also to chemical differences in the several parts. Indeed, *the cord consists, like the brain, of a number of different areas or systems*, each of which, although it may be histologically identical with others, yet follows a special type of evolution in the fœtus, has different connections with more peripheral and more central parts, may possibly be chemically quite distinct from its surroundings, and lastly possesses peculiar pathological predispositions or proclivities which are not shared by contiguous parts, however closely connected with them in anatomical position, and however similar in histological elements.

An apt illustration of these peculiarities is afforded by the manner in which different poisons act on the different portions of the cord. Bread contaminated with ergot of rye will, when habitually taken for some time, cause well-marked disease of the posterior columns; while bread containing an admixture of the *lathyrus cicera*, which is eaten by the lower classes in certain parts of India, Algeria and Italy, will lead to equally striking symptoms of disease of the lateral columns. Strychnia has a special influence in exalting the excitability of the grey centre of the cord, and bromide of potassium diminishes it. Finally, lead will, when absorbed for some time consecutively, cause gradual disintegration of the large ganglionic cells of the grey anterior cornua, and thus lead to a peculiar form of muscular atrophy.

In order, therefore, to render the more minute pathology of the diseases under consideration thoroughly intelligible, it will be necessary for me to enter first into some explanation of the manner in which the highly complex structure of the spinal cord is built up and arranged. Histology and experimental physiology have done very little for the elucidation of this subject, while the study of evolution on the one hand, and of the finer pathological anatomy of the degenerations to which the organ is liable on the other hand, have been of the greatest service for unravelling the intimate structure and arrangement of the organ. Either of these methods of inquiry may be used for controlling the results given by the other, thus affording a greater degree of reliability than each one singly could do.

With regard to evolution, Flechsig[1] has shown that the central nerve-fibre is at first laid down as a naked axis-cylinder, and that this latter is only much later furnished with a sheath of myeline. The latter is therefore a secondary formation, and is, in the several portions of the cord, developed at totally different periods of fœtal life, just as well as the axis-cylinder itself. This mode of evolution follows a definite law with regard to time ; and it may thus be understood why tracts of fibres which are, anatomically speaking, at a considerable distance from each other, are, in fact, far more closely related than others which may be much nearer neighbours.

---

[1] "Die Leitungsbahnen im Gehirn und Rückenmark des Menschen." Leipzig, 1876.

The succession in which the evolution of the different portions of the cord takes place, is as follows :—

1st. The fundamental strands of the anterior columns ;
2nd. The postero-external or Burdach's columns ;
3rd. The anterior mixed zone of the lateral columns ;
4th. The lateral terminal layer of grey matter ;
5th. The postero-internal or Goll's columns ;
6th. The direct cerebellar columns ; and, finally,
7th. The crossed pyramidal strands.

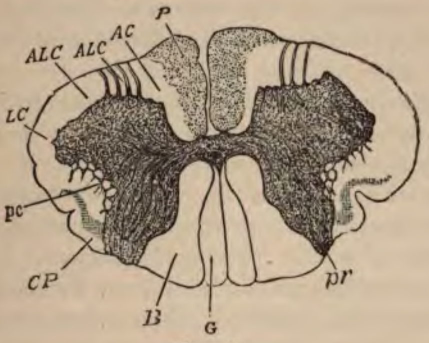

FIG. 2.

Section of the spinal cord from the region of the 5th cervical nerve from a fœtus 12 centimètres long (after Flechsig). *P*, Türck's anterior pyramidal column; *AC*, fundamental tracts of the anterior columns; *ALC*, anterior mixed lateral column; *pc*, processus reticulares; *CP*, crossed pyramidal columns; *B*, Burdach's columns; *G*, Goll's columns.

Of these seven different strands of the cord, those numbered 1, 2, and 3, viz., the fundamental strands of the anterior columns, Burdach's columns, and the anterior zone of the lateral columns, become developed at the earliest period of fœtal life. The naked axis-cylinders are laid down at the end of the fourth week, and their myeline sheaths appear to be fully formed about the end of the fifth month, so that about four months are required for their completion. These three strands have another feature in common, viz., that their transverse section differs at different levels of the cord, being greatest in the cervical enlargement. The size of the transverse section is proportionate to the number of nerve-fibres which enter the strands. From this it is concluded that a large portion of the longitudinal fibres contained within these strands do not remain in the white matter of the cord, but leave it, after a longer or shorter course within the same, in order to proceed into the grey matter of the organ, where they terminate. They therefore connect, on the one hand, the grey matter of the cord with peripheral terminal organs, and on the other hand, different levels of the cord with each other.

Burdach's or *the postero-external columns* (*B*, Fig. 2) are of chief interest for us here, as they appear to be principally selected by the morbid process in tabes. These columns are the bandelettes externes, or rubans externes, or zones radiculaires postérieures (posterior root-zones) of the French anatomists. The term "Burdach's columns," however, is the most convenient and the most generally accepted.

The transverse section of these columns shows great variations. In the cervical enlargement they are more than twice the size of what they are in the middle dorsal portion, and in the lumbar enlargement only two-thirds of it. Their upper portion terminates in the nuclei of the funiculi cuneati of the medulla oblongata. They are the direct continuations of the posterior root-fibres, and therefore establish a direct and immediate connection of the cord with peripheral parts, and thereby with external influences. They are short conducting paths, inasmuch as the posterior root-fibres, which enter into them throughout their course, traverse them in a horizontal direction, or bend upwards or downwards to assume a longitudinal direction. When, therefore, at a given level of the cord numerous root-fibres enter, the calibre of the columns must be greater than where the root fibres are few. On the median side of the posterior horns numerous fibres proceed from Burdach's columns into the grey matter of the cord, in a direction partly towards the posterior commissure, partly towards the posterior cornua, and partly towards Clarke's columns.

These parts contain fibres which connect different levels of the grey matter with each other, and others which proceed into the medulla oblongata. Most of them appear to terminate in the ganglionic cells of the formatio reticularis of the bulb, while another part communicates with the nuclei of the caudate columns and the corpora olivaria.

The French school of anatomists, as represented chiefly by Charcot, Vulpian, and Pierret, have long taught that in tabes the morbid process begins in Burdach's columns, and that the internal portions of the posterior columns are only affected much later on, when the disease spreads to other parts. This proposition has for some time past been generally accepted; but more recent researches by German anatomists, more especially by Strümpell, of Leipzig, have shown that such is not by any means invariably the case; but that, at least in a certain number of instances, the most internal portion of Goll's columns becomes sclerosed in the very commencement of the disease.

No definite law has as yet been formulated with regard to No. 4, the lateral terminal layer of the grey matter.

The postero-internal, or posterior median, or *Goll's columns* (*G*, Figs. 1 and 2), are laid down at the end of the second month, and the formation of their myeline sheaths appears to be finished at the end of the sixth month, giving again a period of four months for the entire process. These strands may only be clearly traced separately in the cervical and upper dorsal portions of the cord, and cannot be easily followed lower down. Their transverse section increases in diameter in a direction from below upwards, and they do not give fibres to the grey matter of the cord, but receive them from it; indeed their origin is in the grey matter of the cord. These fibres proceed from the internal aspect of the posterior cornua, and more especially from Clarke's columns and their immediate neighbourhood, and the posterior commissure. Although Goll's columns are only well seen separate in the cervical cord, there can be little doubt that they course all the way from the medulla oblongata to the lumbar enlargement, where their size is smallest. They terminate upwards in the nuclei of the funiculi graciles, and have to be looked upon as a special system of fibres on account of the quality and calibre of the nerve-tubes composing them, of their origin in the grey matter, and their relation to certain nuclei in the medulla oblongata. While Burdach's columns are short conducting paths, Goll's columns must be considered long conducting paths, intended to connect certain extra-medullary centres in the brain and cerebellum with physiologically identical fibre-systems at different levels of the cord.

The next system of fibres is called the *direct cerebellar strands* (*Ce*, Figs. 1 and 2). They are laid down at the commencement of the third month, while fully formed myeline-sheaths are first observed at the beginning of the seventh month, making again a period of four months taken up for their completion. They contain fibres establishing a connexion between the grey matter of the cord—probably more particularly Clarke's vesicular columns —and cerebellar centres. The fibres of these strands are shown to constitute a special system by the following points:—They spread equally in the grey matter of the cord, behave equally within the lateral columns, and have an equal course in the medulla oblongata. At the time of birth these strands may be seen sharply defined in the cervical cord, and are quite apart from the other portions of the lateral columns. They show a continuous increase in their transverse section, in a direction from below upwards, and may be traced as compact strands to the upper portion of the lumbar enlargement only.

The last system of fibres which we have to consider (7) is that known as the *crossed pyramidal strands.* (*CP*, Figs. 1 and 2.) These are the latest of all formations.

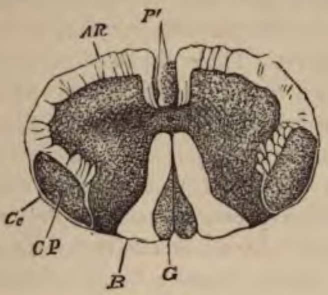

FIG. 3.

Section of the spinal cord from the middle portion of the cervical enlargement of a fœtus, 35 centimètres long (after Flechsig). *P*, anterior pyramidal strand; *AR*, anterior roots; *Ce*, direct cerebellar columns; *CP*, crossed pyramidal strands; *B*, Burdach's columns; *G*, Goll's columns. Magnified 6·4 lin.

They are first laid down about the middle of the fifth month, and the formation of their myeline-sheaths is only finished about the end of the ninth month, so that again a period of four months is seen to elapse between their beginning and their eventual completion. These strands contain exclusively fibres which are in connection with the cerebral hemispheres, through the pyramids of the medulla oblongata and the higher motor tracts; and they communicate with certain accumulations of grey matter in the brain, more especially the lenticular nucleus and the central convolutions of the cortex.

In other words, they contain all the fibres which form a direct connection between the grey matter of the cord, the central ganglia, and the motor portion of the cortex of the brain. They therefore constitute likewise a special system of fibres, having its own definite peculiarities and terminations. They have this feature in common with the direct cerebellar strands, that there is a steady increase in the diameter of their transverse section in a direction from below upwards. They are situated in the posterior half of the lateral columns, and may be traced down as far as the lower end of the lumbar enlargement, or to the third or fourth sacral nerve. They diminish in transverse section from above downwards, owing to their fibres successively entering the grey centre of the cord in that portion of it which connects the anterior and posterior cornua. They show immense individual variations in extent, inasmuch as the fibres which proceed from the motor region of the brain into the cord have the choice to course either in the same anterior, or the opposite lateral, column, before they proceed into the central grey matter.

From the foregoing description it appears that *the different conducting paths in the spinal cord are laid down and developed in an absolutely systematic manner*, and that at the same period of intra-uterine existence only one or several allied systems are formed. Those which are most important for the life of the fœtus and which are reflectory by nature, are early developed, their faculty of incessant function being an indispensable condition for viability altogether. On the other hand, those tracts which place the spinal cord under the influence of the psychomotor centres, or the will, become evolved at a later period. These latter are more abundantly developed in man than in the lower animals, and are not formed at all in congenital absence of the brain, or in destructive disease of the motor area of the same.

As far as size is concerned, the anterior columns have been found to contain on the average 18 per cent., the lateral columns 46·6, and the posterior columns 35·4 per cent. of the white matter of the cord.

Anomalies of anatomical growth have probably a considerable influence on the evolution of diseases which occur later in life. They may possibly be found eventually to be *the chief material base of what is now called the neurotic constitution.* Congenital fissures or cavities may be the starting-point of certain diseases, causing tendency to the formation of glioma, glio-sarcoma, or chronic interstitial or parenchymatous degeneration. Singular irregularities in the distribution of the white and grey matter are not rarely met with in persons who have died of diseases of the nervous system. Schultze has found such anomalies in cases of spastic paralysis and general paralysis of the insane. Kahler and Pick discovered, in a person who had died of tabes, abnormal smallness and irregular shape of the central grey matter, and extreme smallness of the posterior columns, which was not exclusively owing to degeneration. As there are born statesmen, artists, and criminals, so there are no doubt some born to be sclerosed, and these are in general the descendants of the syphilitic, the gouty, and the alcoholised. The gradual formation of the myeline sheaths of the central nerve-fibre, which has been shown to follow a definite law throughout embryonic life, may in some cases be carried out in a deficient manner, and this may later on lead to disease of the imperfectly formed or not fully protected parts. Where undue claims are made upon a small or badly formed medulla oblongata or spinal cord, where connective tissue predominates over nerve-cells or fibres, there must be greater tendency to the development of asthma, diabetes, tabes, or paralysis, than there will be in cases where the essential constituents of these organs have been more abundantly and perfectly developed.

CHAPTER II.

MORBID ANATOMY OF TABES SPINALIS.

THE post-mortem appearances in tabes have been studied by a very large number of observers, and the main facts connected with them appear now to be tolerably well ascertained, although opinions still differ widely with regard to their interpretation.

1. With regard to the *spinal membranes*, it is found that the dura mater is habitually normal, while the arachnoid may be opaque, and the pia mater is frequently congested and thickened at the level of the posterior columns. The spinal fluid is increased, and sometimes to a considerable extent. The trabeculæ which traverse the sub-arachnoid space may be thickened and more abundant than in the normal condition. The thickening of the membrane and the hyperæmia of the vessels of the pia mater become gradually less towards the region of the lateral columns, and are in the majority of cases more pronounced in the dorso-lumbar than in the cervical region of the cord. *Posterior spinal lepto-meningitis* has, therefore, to be looked upon as an habitual concomitant of tabes, more especially where the disease is seen in an advanced stage.

2. The most constant, most decided, and earliest change, however, is found in the *posterior columns* of the cord, which show a definite lesion recognizable by the microscope at a time when the clinical symptoms have only commenced to manifest themselves, and when the parts appear perfectly normal to the naked eye. Where tabes has lasted for years, as is usually the case, the unassisted

eye recognises the appearances of grey induration, or sclerosis in the posterior columns; that is, these parts appear flattened and reduced in their transverse diameter, while their colour has changed from white to grey. In some cases the discoloration is more pinkish, reddish, or yellowish. This alteration is generally more marked in the lower and middle portions of the cord, and diminishes gradually in the direction towards the medulla oblongata, on the one hand, and the cauda equina on the other hand. The tissue of the posterior columns is firmer and tougher than in health, and really sclerosed; but in exceptional cases the consistency is normal, or may appear even softer than usual. These differences are owing to the variations in the overgrowth of connective issue, which is in the majority of cases very marked, but in some less perceptible. As a rule, the degeneration does not spread beyond the medulla oblongata, but occasionally a certain amount of corresponding change has been seen in the superficial layers of the pons varolii and the corpora quadrigemina.

The real nature of the morbid alteration which has taken place is only revealed after hardening and staining the cord in various fluids and by microscopic examination of fine sections. Gerlach and Lockhart Clarke were the first to introduce the process of hardening the organ by chrome, and used for this purpose a 0·25 per cent. solution of crystallized chromic acid. It was, however, soon discovered that this does not harden the parts so thoroughly and uniformly as the chrome-salts. One of the most useful preparations of the latter kind is Müller's fluid, which consists of one part of sodium sulphate and two and a half parts of potassium bichromate in a hundred parts of water. By immersion into this fluid, the cord is not only hardened, and therefore better suited for section, but the diseased parts are rendered more clearly perceptible by remaining light coloured, while the healthy parts absorb the chrome-salt readily, and therefore assume a darker colour. Another excellent agent for hardening and staining the cord is a one per cent. solution of osmic acid, as recommended by Exner, of Vienna. In France this method is generally described as Ranvier's. Osmic acid has the peculiarity of deeply staining the myeline sheath of the central nerve-fibre, which appears blackened, but has no effect on the axis-cylinder. It is therefore well supplemented by a solution of aniline blue-black, in the proportion of one to four hundred parts of water, which has been introduced by Bevan Lewis. This latter fluid has just the opposite effect, since it leaves the medullary sheath unaltered, but imparts a deep blackish stain to the axis-cylinder. Charcot and Vulpian employ chiefly an ammoniacal solution of carmine, which stains the grey matter more deeply than the white, and in the white matter only the connective tissue, and not the nerve-tubes. A pink coloration of the white columns of the cord therefore shows, even to the naked eye, the presence of sclerosis, the tint being deeper in proportion to the intensity of the degenerative changes. Other useful staining fluids are Ranvier's solution of picro-carmine, and the compound dyes of picro-aniline, hæmatoxyline with aniline, picro-carmine with iodine green, nigrosine, fuchsine, and eocine.

However valuable these several methods of hardening and staining the cord are, we must yet not lose sight of the fact that they involve a great expenditure of time, are often unsatisfactory in the hands of less practised observers, and modify the structure of the parts considerably by corrugation. In order to avoid these drawbacks, histologists have for some time past resorted to freezing the fresh structures, thereby avoiding any chemical alteration. For some years the ice-and-salt freezing microtome, as perfected by Rutherford and Williams, was the one principally employed ; but this process is objectionable, inasmuch as the degree of freezing cannot be well regulated. Excessive refrigeration causes the crystals of ice which are formed to break up the delicate nervous tissue, and therefore spoils it somewhat for examination. The ether-freezing microtome, as introduced by Bevan Lewis,[1] therefore constitutes a real progress. In the construction of this instrument, Richardson's spray-producer has been ingeniously utilised, with the result that the freezing can be checked at any stage, and rene wedwhen required. The structures, therefore, do not assume that hard icy consistence which blunts or turns the edge of the blade, but may be made to acquire just such a degree of density as is best suitable for section-cutting.

Another process, which is chiefly suitable for examining the ganglionic cells of the grey matter of the cord in the fresh state, is that known as dissociation, which was first systematically practised by Gerlach, and afterwards perfected by Bevan Lewis. In the latter method, sections are placed in Müller's fluid for a few minutes, and then gradually compressed by a mounted needle between cover-glass and slide, when the large ganglionic cells will easily come in view.

With all these different methods of microscopic examination at our disposal, however, it must be confessed that, although we may, and undoubtedly do, gain a thorough insight into the localisation of the morbid processes, the more minute alterations of the nervous matter which take place in disease still escape inquiry to some extent, as the very means used for examining the structures produce a mechanical or chemical alteration of the diseased parts, and thus add an artificial element which it has hitherto been impossible to eliminate.

In advanced stages of tabes, the microscopic examination of the parts shows that the medullary sheath as well as the axis-cylinder of the nerve-tubes constituting the posterior

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[1] "The Human Brain," p. 92. London, 1882.

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columns have disappeared. Their place is taken by a loose areolar tissue, the meshes of which contain fluid during life, and, after staining, the different substances used for that purpose. A few healthy nerve-fibres are generally found lying scattered about in this tissue, while others may be seen in various stages of degeneration. They appear granular, varicose, and narrower than in health. Various degrees of hyperplasia and fibrillary metamorphosis of the connective tissue which cements the central nerve-fibres are almost invariably present. Only few cells and nuclei are found in the overgrown neuroglia, which is accounted for by the slow progress of the morbid alteration, the cells thus growing old and becoming gradually transformed into a firm fibrillary tissue ; but numerous amyloid bodies are interspersed in the mass.

The arterioles of the posterior columns are likewise found to have undergone a marked change. The adventitia is thickened and studded with oil-globules and granular pigmentary corpuscles. These latter formations are also seen in the lymphatic spaces between the external and middle tunic, and even in the tiniest capillaries, whose coats are reduced to endothelium. The arterioles appear as whitish lines on the grey groundwork of the wasted columns, proceeding in a longitudinal direction. Innumerable amyloid bodies are met with along the course of the arterioles ; and these are chiefly seen where the degeneration is not very far advanced, while they are less abundant after the nervous matter has been entirely destroyed.

What region of the posterior columns is first affected? In general the change appears to commence, and is certainly most marked, in the lower dorsal and upper lumbar portion of the cord, while the lower lumbar and cervical portions are less affected. Yet, even in recent cases, the cord appears to be generally affected in its entire extent, from the medulla oblongata downwards. In cases of long standing the entire transverse section of the posterior columns, including both Goll's and Burdach's columns, is sclerosed, while in the earlier stages of the disease the process is more strictly localised in certain areas.

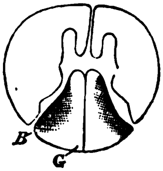

FIG. 4.

Section of cord in which Burdach's columns (*B*) are sclerosed and Goll's columns (*G*) healthy.

Pierret, who was the first to examine this point more minutely, concluded from his observations that the disease commenced with two symmetrical islands of degeneration in Burdach's columns, while Goll's columns only became affected at a subsequent stage of the malady with a kind of secondary degeneration. This view was endorsed by Charcot and Vulpian, and was for some time unreservedly accepted by the profession; but more recently Strümpell,[1] of Leipzig, has again carefully examined this point, and

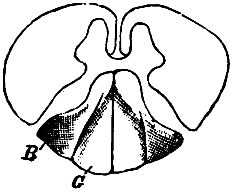

FIG. 5.

Section of cord in which Burdach's columns (*B*) and the external portion of Goll's columns (*G*) are sclerosed.

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[1] "Archiv für Psychiatrie," p. 749. Berlin, 1883.

has arrived at some novel results of importance, whereby Pierret's conclusions have been considerably modified.

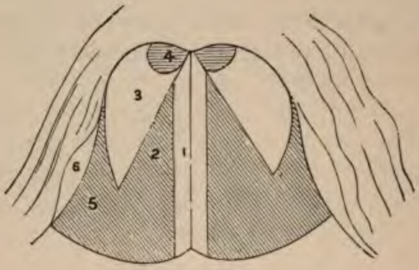

FIG. 6.

Schematic diagram of the different areas of degeneration in the posterior columns, after Strümpell. 1. Small portion of diseased tissue at each side of the posterior commissure. 2. The bulk of Goll's columns converging anteriorly in wedge-shaped fashion. 3. Antero-lateral area of Burdach's columns, with point directed backwards. 4. Small round field at the anterior broad extremity of the lateral anterior area, and sharply separated from the apex of Goll's columns. 5. Postero-external field, with point directed forwards. 6. Small band of tissue at inner side of posterior roots.

According to Strümpell, appearances differ in the early stages of tabes in the different portions of the cord. In its dorsal portion, two small antero-lateral areas (Fig. 6, No. 3) are first affected, and this is the part from where fibres proceed to the posterior cornua ; but almost at the same time a similar degeneration appears in a small median zone along the posterior fissure (Fig. 6, No. 1) at the innermost portion of Goll's columns. Further on in the course of the disease the bulk of Goll's columns (Fig. 6, No. 2) begin to suffer, and their posterior portions more than their anterior ones. The postero-external field of Burdach's columns (Fig. 6, No. 5) remains normal for a long time ; but in advanced cases the entire mass of the posterior columns is found affected.

In the lumbar portion of the cord, the disease commences in the middle area of the posterior root-zone, while its anterior and posterior part remain healthy for a long time. Later on the posterior area degenerates likewise, and only a small field near the posterior fissure remains spared. The anterior zone remains much longer normal, and appears in cases of typical tabes to escape altogether.

In the cervical cord there are two small lateral areas of degeneration (Fig. 6, No. 4), which are broader anteriorly. Goll's columns appear to be affected in the beginning. After a time the posterior root-zone becomes affected, but the antero-lateral section (Fig. 6, No. 3) and the postero-external field (Fig. 6, No. 5) resist the inroads of the disease for a considerable time.

According to these researches, therefore, matters appear to be even more complicated than they seemed previously. Goll's columns are shown to consist of three separate divisions, viz., first, of a thin strand of fibres which are most interiorly situated, close to the sides of the posterior fissure, and which are shown to have a special position in the system by degenerating independently at an early stage of the disease ; secondly, of the real bulk of Goll's columns, which are wedge-shaped and pointed anteriorly ; and, thirdly, of the small round anterior field (Fig. 6, No. 4), which appears to be sharply separated from the contiguous apex of Goll's columns.

3. The *posterior nerve-roots* are generally found wasted, and in old cases so much so that it may be difficult to discover them, more especially in the dorso-lumbar portion of the cord, where in general the change is greatest. They show a grey, reddish or black discoloration, and form a striking contrast to the anterior roots, which remain white, large, and plump. On examining the posterior roots, it appears that the nerve-fibres are destroyed, having undergone granular and fatty degeneration ; and this destructive process invades more particularly the axis-cylinder, while

the medullary sheath resists much longer, and may still be seen after everything else is gone. If nerve-fibres are still met with in advanced cases, their diameter is greatly reduced.

4. The *spinal ganglia* show, as a rule, no alteration whatever, not even those which correspond in anatomical position to the most severely affected areas of the posterior roots and columns. Vulpian has found these structures sometimes perhaps a little more pigmented than usual; yet it should be considered that healthy cells often contain a good deal of pigment. In general the nucleus and nucleolus of the cell does not appear to be altered. Luys and Pierret have in a few cases seen some amount of wasting in these ganglionic masses, but this is certainly a very exceptional occurrence. In connexion with this point it should not be forgotten that our means for ascertaining the exact condition of ganglionic cells are still defective, and that it is most difficult to demonstrate satisfactorily that there is any real atrophy in these structures.

5. *The central grey matter of the cord*, on the other hand, appears not unfrequently to participate more or less in the disease. It is more especially the posterior cornua, and the place of junction between the anterior and posterior cornua, and Clarke's vesicular columns, which are often found affected. The sclerosis, however, appears to invade more the nerve-tubes and the neuroglia than the ganglionic cells, and there can be little doubt that the same fibres which become destroyed in the posterior columns, waste in their further course in the grey matter. Where, therefore, the posterior cornua are found reduced in size, this is owing to atrophy, not of cells, but of fibres. The anterior cornua, with their giant cells, have mostly been found healthy; yet Leyden[1] has recently drawn attention to a peculiar

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[1] "Tabes Dorsalis," p. 15. Vienna, 1883.

condition of these cells, more especially in the lumbar enlargement of the cord, which appears to be by no means unfrequent. There is no actual wasting in them; but they appear strongly pigmented, tough, harder and rounder than usual, more or less reduced in size; and their processes are small, tough, and fragile. It seems not unlikely that this may be the anatomical substratum of the flabby, weakened, and ill-nourished condition which is habitually found in the muscles of tabid patients, more especially in the advanced stages of the disease. This is, however, entirely different from actual wasting of the muscular fibre, which also occurs not unfrequently in tabes. I have notes of a somewhat considerable number of cases in which sometimes early, sometimes late, the well-known clinical signs of muscular atrophy—*i.e.*, destructive wasting of fibres, with fibrillary twitches in the affected muscles—were added to the symptoms of tabes.

There can be no doubt that this is not a simple coincidence, but that there exists a peculiar connection between sclerosis of the posterior columns and atrophy of the anterior cornua. The muscular atrophy observed in tabid patients has not the well-known distribution of progressive muscular atrophy, or of lead-palsy. It is in general limited to certain regions, for instance the hand, where it affects the balls of the thumb and the little finger, as well as the interosseous muscles; or it may affect the foot, certain portions of the back and the neck, and, after existing for some time in one side, it will eventually attack the symmetrical portion of the other side. I have at the present time a patient under my care in whom the symptoms of tabes have affected principally, although not exclusively, the left side of the body, and where there is marked wasting of all the muscles of the left hand. The muscles of the forearm, arm, and shoulder do not show the slightest signs of atrophy. On the other hand, fibrillary twitches have just commenced to occur in the interosseous muscles of the right hand, with a corresponding amount of debility, showing that the affection is beginning to invade the symmetrical portion of the other side.

Cases of this kind have but rarely come on the post-mortem table; but Pierret[1] has recorded one in which there had been well-marked wasting of the muscles of the right side of the body during life, and where after death the entire anterior horn of the right side of the cord was found to be wasted, while the left anterior horn had remained normal. The diameter of the right horn was much less in the cervical portion than that of the left; groups of myelocytes were seen close to the giant cells; the latter showed a large accumulation of pigment and commencement of atrophy. In the cord, Clarke's vesicular columns showed considerable alterations; some of their cells had entirely disappeared, others were evidently diseased and were only represented by small heaps of brown granulations. The right anterior horn had only about half the dimension of the left, and some of its cells had undergone diverse degrees of pigmentary atrophy. In the lumbar region this lesion was much more marked. Some of the giant cells had here completely disappeared, and their place was taken by a finely granular fibrous tissue containing oil-globules and amyloid bodies. The shrinking of this tissue had no doubt to a great extent caused the diminution in the diameter of the right anterior horn. To this corresponded certain appearances in the affected muscles of the limbs and body, likewise on the right side. They were pale and thin; there were a few primitive bundles affected with granular degeneration, while there were numerous bundles which showed simple atrophy, yet had preserved their transverse stripes. The nuclei of the sarcolemma were greatly proliferated, causing some of these bundles actually to be distended by them. These changes corresponded in degree exactly to the changes in the anterior horns, being more marked in the muscles of the lower extremity than in those of the body and the upper limb.

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[1] "Archives de Physiologie," p. 599. Paris, 1871.

Bevan Lewis[1] has lately described a case of tabes, in which the posterior columns appeared to be almost destroyed in the dorsal portion of the cord; and where in those regions which were stamped by the greatest intensity of morbid change, the anterior cornua had invariably suffered, the various groups of cells being either greatly wasted or wholly absent; and the hemispheric distribution of the lesion was such that, whilst one horn might be entirely free from disease, the opposite one might be found shrunken to one-half its proper dimensions. This atrophy was most apparent in the central and lateral groups of the cells. In that case the direct cerebellar column was also involved, while the crossed pyramidal, and Türck's columns were perfectly healthy. The anterior root-zone, however, showed a morbid change limited to one hemisphere, and probably consequent upon changes in the corresponding cornu. Singularly enough, no muscular atrophy is mentioned as having existed during life in the clinical history of the case, but this may possibly have been overlooked.

6. The *lateral columns* of the cord are found to be normal in the earlier stages of the disease, but may become affected later on. It might be assumed that this arose from a simple spreading of the degeneration *in loco*, and in a transverse direction, as in myelitis; but it is more probably an independent system-disease, which becomes complicated with tabes in its more advanced stages. This view seems to me the more probable one, more especially as we find that the lateral column is not diseased *in toto*, but that chiefly that portion of it is affected which is known as Flechsig's direct cerebellar column. In ex-

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[1] "Brain," January, 1884.

ceptional cases, however, the affection has been seen to spread as far as the anterior columns.

7. The *sympathetic system of nerves* appears to be generally unaltered. In a few cases, however, wasting of the myeline-sheaths and of the nerve-cells of the sympathetic ganglia has been noticed. Recent observations to this effect have been made, under the superintendence of Vulpian, by Raymond and Arthaud, who found proliferation of connective tissue, thickening of blood-vessels, atrophy of ganglionic cells, oil-globules with colouring matter, and fatty degeneration of the nuclei of Remak's fibres.

8. In the *brain* there may be no change whatever. As a rule the affection does not extend beyond the bulb, where the nuclei of the fifth and auditory nerves are occasionally seen sclerosed. Where, however, tabes ultimately becomes complicated with epilepsy, general paralysis of the insane, and other analogous conditions, diffuse changes in the cortex and the pia mater of the brain have been discovered.

9. The *cranial nerves* are not unfrequently found affected. The *olfactory* nerve is liable to atrophy; and its sheath may be filled up with amyloid bodies. This wasting appears to affect chiefly the external root, which may be traced to the fissure of Sylvius, and which is more important for olfaction than the middle or internal root of the nerve. Some time ago a patient was under my care at the hospital, in whom there had been symptoms of acute olfactory neuritis followed by complete anosmia in the initial stage of tabes, and who died of sudden collapse eight years afterwards. Wasting of the first pair of nerves at the base of the brain was discovered *post mortem*.

The *optic nerve* suffers habitually in tabes, and often quite in the beginning of the malady. It has been found reduced to one-half or even one-third of its ordinary diameter, and shows to the naked eye the same state of grey induration as is seen in the posterior columns of the cord. The axis-cylinder and medullary sheath are wasted, and a tough mass of overgrown neuroglia is left, with which is mixed amorphous matter, some oil-globules, and amyloid bodies. Occasionally a few healthy nerve-fibres may be left. The papilla of the nerve is whiter and more depressed than in health, and the arterioles are less visible. The disease is generally believed to begin in the peripheral end of the nerve, and from there to creep further on to the centre. If both nerves are affected, the wasting is found to extend backwards to the chiasma, the optic tracts, and the corpora geniculata. When only one nerve is diseased, the chiasma loses its shape, and of the two optic tracts the one opposite to the diseased nerve is sclerosed, while the one on the same side is healthy, showing that there is crossing of fibres in the chiasma.

While, therefore, until now it has been generally held that optic atrophy begins in the peripheral expansion of the nerve, Poucet has lately come to the conclusion that the alteration is more central than peripheral. He examined a case of tabes where the patient had been blind for ten years, and discovered sclerosis of the optic nerve in its orbital portion, and atrophy without sclerosis in its cerebral portion. In the retina, there was destruction of the proper fibres of the optic nerve and the ganglionic cells, while the internal and external granular layer (the latter identical with Ranvier's "visual cells") was healthy. In the region of the macula, the cones were healthy. Poucet therefore considers that in the blindness of tabes the alteration is not peripheral, but central; that the sclerosis of the orbital portion of the optic nerve is secondary to the parenchymatous atrophy, and that the tissue and cells of the neuroglia show no overgrowth. Further observations on this point are highly desirable.

The *third* nerve has been found wasted, either in its entirety or in some of its branches; and I have seen the same condition in the *sixth* nerve in a case where the patient had suffered from persistent paralysis of the external rectus muscle.

The nuclei of the *fifth* and *auditory* nerves have been found wasted by Hayem and Pierret; but whether these nerves suffer in their peripheral course is as yet uncertain. As they draw their trophic influence not only from their nuclei in the medulla oblongata, but also from ganglionic cells with which they communicate further in front, it does not absolutely follow that sclerosis of their nuclei must needs entail wasting of the peripheral expansions of the nerves. Some time ago I had a case under my care at the hospital in which a patient suffering from tabes had become completely deaf in consequence of what appeared to have been an attack of acute double auditory neuritis. The patient recovered from all symptoms of tabes, but remained stone-deaf; and were it to come to an inspection, I have no doubt that complete atrophy of both auditory nerves would be discovered in that case.

It will be important, in future investigations of this kind, to keep in mind that the auditory nerve has been physiologically and histologically proved to be a compound nerve, and to consist of two different portions, one of which is the nerve of the special sense of hearing, while the other is the nerve of the sense of space, and supplies the semicircular canals, which are the peripheral organs of the sense of space. During their transit through the internal auditory meatus, these two nerves are perfectly distinct, owing to a well-developed strand of connective tissue between them. The finer histology of these nerves has recently been studied by Erlitzky, Axel Key, and Retzius. According to these observers, the nerve consists of a larger anterior and inferior, and a smaller posterior and superior portion. These two portions are distinguished by the characters of the nerve-tubes composing them. In the former portion dissociation shows delicate fibres, whose axis-cylinders are only slightly coloured by carmine, and which are covered by a very slight circular layer of myeline; there are no nuclei in Schwann's sheath, nor annular constrictions; the fibres show frequent enlargements all along their course, probably owing to the axis-cylinders and their sheaths, while the layer of myeline shows the same thickness in the enlargements as in the intermediate portions. This is the *cochleary* or true auditory nerve. The second portion contains much thicker fibres, which are well coloured by carmine, and show annular constrictions. They resemble the other nerves, and constitute the *vestibulary* nerve, or the nerve of space. Of these two portions of the nerve, the space-fibres appear to suffer much more frequently in tabes than the auditory fibres, more especially in the earlier stages of the malady, and should, therefore, be examined with particular care.

The nuclei of the *glosso-pharyngeal* and *vago-accessory* nerves have likewise been found sclerosed. The roots of these nerves form, together with sympathetic fibres, a kind of intermediary system with motor and sensory zones. This corresponds to Stilling's "solitary strand," and to Clarke's "slender column," gives off the intermediate nerve of Wrisberg, which is vasomotor, and is connected with motor and sensory ganglia. This strand proceeds at the level of the decussation of the pyramids towards the side of the spinal accessory nerve, and is found in the upper portion of the cord in the intermedio-lateral tract, whence sympathetic fibres take their origin. Pierret believes that sclerosis of this tract is the cause of the gastric crises and other vasomotor and visceral troubles which occur in tabes. Kahler has quite recently described a case in which there had been pharyngeal paralysis, aphonia from loss of power in the right vocal cord, fits of spasmodic cough, and gastric crises. At the necropsy, the oblongata was found healthy, with the exception of sub-ependymal sclerosis, which penetrated into the grey matter on the floor of the fourth ventricle, and involved the nuclei of the vago-accessory nerves, more especially the one on the right side.

10. The *spinal nerves* and their terminations in the skin, the joints, the muscles, etc., have generally been believed to remain healthy in tabes. In his latest essay on the disease, in Eulenburg's "Encyclopädie der medicinischen Wissenschaften" (Vienna, 1883), Leyden says that no alterations in these nerves have been discovered. Yet there can be little doubt that disease of these parts has been frequently overlooked, partly from preconceived ideas about the nature of the malady, and partly because to the naked eye these nerves may appear perfectly healthy, showing neither softening nor induration, œdema, or any other change. Yet it had often struck observers, in examining cases of tabes clinically and anatomically, that there was no apparent relation between the symptoms in the sphere of sensibility observed during life and the lesions found in the posterior columns and nerve-roots after death. This naturally led to the supposition that there might be, apart from the central lesion, likewise a peripheral one in the cutaneous nerves.

Langenbuch, when proposing the operation of nerve-stretching for the relief or cure of tabes, described certain gross lesions which he had perceived in the large nerves of the extremities after they had been laid bare for the purpose of operation. Subsequent writers, however, stated that such lesions did not exist, but had been found necessary in order to render the wonderful effects of nerve-stretching more intelligible! Such a state of opinion makes it incumbent upon us to consider this question somewhat more in detail than we should otherwise have considered necessary.

Türck, of Vienna, who was the first to describe secondary sclerosis of the lateral columns of the cord, consequent upon cerebral hæmorrhage, and whose keen insight into the morbid anatomy of the nervous centres altogether was very much in advance of his time, took up this inquiry as far back as 1858, but was led to negative results, as was also Vulpian in 1868. Westphal was the first to discover, ten years later, atrophy of the cutaneous branches of the crural nerve in a case of combined posterior and lateral sclerosis. Pierret afterwards expressed his belief that the lightning pains, the anæsthesia, and certain trophic disturbances in tabes depended upon a form of peripheral neuritis analogous to optic neuritis, which was seen in the terminal expansions of the cutaneous nerves, became less marked further away from the periphery, but was again discovered in the central ends of the affected nerves.

Pitres and Vaillard[1] have recently made a very able inquiry into this point, and found that in certain cases of tabes histological alterations occur in the peripheral branches of the spinal nerves resembling very closely those which are known as Wallerian degeneration, viz., such as occur after experimental section in the divided nerve. In the beginning there is a swelling of the nucleus of the fibres and segmentation of myeline; and eventually complete destruction of the axis-cylinder and the medullary sheath takes place. This form of neuritis has, however, not the uniform course of Wallerian degeneration, but is sometimes slow and at other times exceedingly rapid in its progress. The ordinary Wallerian degeneration follows a regular evolution, the phases of which take place at perfectly definite intervals, and with a kind of mathematical certainty which has been determined by experiment, and which can be exactly foreseen. The form of neuritis, however, which occurs in tabes has no such regular course. It may moreover spread from the periphery to the centre, which never occurs in Wallerian degeneration. The inflammation appears to affect only the nerves, and not the intertubular connective tissue.

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[1] "Archives de Neurologie," Paris, 1883 (March to September).

The latter only appears to overgrow after the neuritis has already destroyed the nerve-tubes. The course of the neuritis may either be very rapid or slow; and while there is an ascending centripetal tendency, yet there does not appear to be any continuous alteration between the cord and the altered nerves, the nerve-trunks having generally been found healthy.

Pitres and Vaillard have described five different types of inflammation, viz., fragmentation of myeline (1) into shafts, (2) globules, (3) granulations, the latter giving the nerve-fibre a varicose aspect; (4) atrophy of tubes with yellow granulations in the interior of the sheaths; and finally (5) complete atrophy with empty sheaths. These different alterations are, however, rarely found to invade the nerve-tubes in exactly the same manner, but seem to combine in a variety of ways. Thus, with fibres whose myeline has been divided into voluminous shafts, one finds varicose and partially atrophied fibres; or varicose tubes are mixed with atrophied tubes containing only yellow granulations. In some cases there were found, by the side of deeply degenerated fibres, delicate nerve-tubes with greyish contours, such as one meets with in embryonic rather than in adult fibres; and these are probably regenerated fibres, resembling those which are seen at the peripheral extremity of divided nerves, three or four months after their section. In the intrafascicular connective tissue the cellular elements were more numerous, swollen, rounded, provided with a large nucleus, their protoplasm being filled with granulations varying in colour from light grey to deep yellow.

It must also be mentioned that these peripheral neuritides do not invariably give rise to nutritive or sensory disturbances. The latter only occur when the proportion of altered nerve-fibres is very considerable, and vary according to the extent of the change and to the special functions of the affected fibres. Thus in one case of tabes, Pitres and Vaillard found that, while the median and musculo-spiral nerve were quite healthy at the lower end of the humerus, there was atrophy in the dorsal branch of the musculo-spiral nerve at the lower portion of the forearm, and in the collateral nerves of the thumb, first and third fingers. The clinical symptoms corresponding to this were deformity of the metacarpo-phalangeal joint of the first finger, the surfaces being swollen and it being easy to dislocate the phalanx on the metacarpal bone as well as to reduce the dislocation afterwards. Nothing particular, however, was noticed in the thumb and first finger. In the lower extremity there was found atrophy of nerve-tubes, chiefly in the plantaris internus, and the collateral internal and external nerves of the big toe; to which during life had corresponded perforating ulcer in the soles of the feet and dystrophy of toe-nails. Further signs were sclerosis of the posterior roots and columns, while the intermediate nerve-trunks and the anterior roots were perfectly healthy. Analogous changes were discovered in cases of Pott's disease and myelitis, owing to caries of cervical vertebræ. In the latter case there were bedsores on the sacrum and heel, pemphigus, and dystrophy of the toe-nails.

It is probable that the central lesion is not the immediate cause of the peripheral neuritis, but only a predisposing condition, and that other influences, such as pressure, etc., are likewise necessary for its production. It is also worthy of remark that this form of neuritis is not peculiar to tabes, but occurs after hemiplegia from cerebral softening, fracture of the skull, in herpes zoster, leukæmia, and in other conditions where the power of the central nervous system is reduced below par. It causes, according to the function of the affected nerve, either severe symptoms in the sphere of sensibility, or various nutritive disturbances, such as vesicular and bullous eruptions, hard œdema, and perforating ulcer, as well as the acute bedsore which sometimes supervenes in the course of certain diseases of the nervous centres, and is probably intimately connected with the occurrence of Charcot's joint-disease. It has no doubt been often overlooked, chiefly because the changes in the nerves are not perceptible to the naked eye, but are only found after staining and by microscopic investigation.

These arguments appear to me to find considerable support in some recent observations of Déjerine, who has described two cases in which there had been during life very marked sensory disturbances of nearly the same degree of intensity, but where the inspection showed in one case extensive disease of the posterior roots and columns, and hardly any such lesion in the other. On examining, however, the nerves in the latter case, neuritis was discovered in their peripheral expansions, while their more central parts, and also the spinal ganglia, which are their trophic centres, were healthy.

Pierret considers it probable that the corpuscula tactus in the skin are primarily altered, and that the peripheral neuritis is a consequence of this alteration; but this view is contradicted by observations of Langerhans, who examined the corpuscula tactus in six tabid patients in whom considerable alterations of cutaneous sensibility had occurred during life, and found them perfectly normal.

The alteration of cutaneous nerves in tabes would therefore appear to be quite independent of their trophic centres and terminal expansions, and to constitute a true peripheral neuritis, which is not necessarily related to the medullary lesion.

Until quite recently alterations of sensibility found to exist in tabid patients have been ascribed to lesions of the posterior columns and roots; and much further research will be necessary before we can be sure that peripheral neuritis is at all constant under such circumstances. There can, however, be no doubt that, when it is discovered, it will explain why cutaneous symptoms should have been prominent, while in cases where it is absent or slight such signs might have been wanting. Neuritis probably plays an important part in the retardation of sensation, and also in the diminution of faradic sensibility, which is so frequently observed in cases of tabes.

11. Charcot's *arthropathies* finally claim our attention. It is not a little singular that this condition, which is not so very rare—for Charcot has seen it to occur on the average in one out of ten cases of tabes—should, until quite recently, have been overlooked; and that the creators and custodians of the great anatomical museums in the different capitals of Europe, such as Hunter, Stanley, and Sir James Paget in London, Johannes Müller in Berlin, Dupuytren in Paris, etc., should not have preserved any specimens of it. The idea has therefore been started that we have here to do with an entirely new disease; but it appears to me much more rational to assume that these lesions have been habitually confounded with those produced by rheumatic gout, and that Charcot's keen clinical instinct, assisted by his unrivalled opportunities of having constantly hundreds of old women in various stages of spinal disease under his eyes at the Salpétrière, succeeded in connecting certain joint-lesions with the occurrence of tabes.

A benign and malignant form of this affection is distinguished. In the former there is a sudden but painless effusion of serum, which is gradually absorbed and causes no further trouble; while in the latter there is rapid destruction of a joint, and dislocation of the head of the bone, which becomes presently eroded and atrophied, without the formation of stalactites or the ordinary appearances of arthritis sicca. If the disease attacks the shafts of the bones, spontaneous fracture may occur from atrophy. In some cases indeed the fragility of bones appears so great that the most trivial causes seem to induce fracture. Thus a sudden movement of the leg has led to fracture of the thigh-bone; and in a case recorded by Von Bruns, a patient actually broke his jaw through munching a lump of sugar! In one case, fractures occurred in six different bones. A marked lesion in such bones is a widening of the Haversian canals, and great deficiency of phosphate of lime.

The pathology of arthròpathies and other trophic changes supervening in the course of tabes, such as spontaneous fracture, perforating ulcer, falling out of nails, hard œdema of the skin, eruptions, etc., is still very obscure. When Charcot[1] first described them (1868-70), the connexion between the integrity of the large ganglionic cells of the anterior cornua and the nutrition of the muscles had just been ascertained. It was then thought that in cases of arthropathy the anterior cornua were at fault, and a few observations seemed at first to confirm this idea; but soon afterwards contradictory facts were observed, and that theory has now been given up. Moreover it would be difficult to understand why arthropathies should not be common in infantile paralysis or progressive muscular atrophy, where the anterior cornua are notoriously affected. On the other hand, the joint-affection is not habitually connected with wasting of the muscles. It seems, therefore, much more probable that the arthropathy is owing to local changes in the peripheral nerves. The nerves of the joints and the muscles may undergo neuritis, as seen by Pitres and Vaillard in a case of tabes, where the left sciatic nerve, as well as the left posterior articular nerve and the muscular nerve of the same side showed degeneration. These changes corresponded to the following symptoms observed during life:— The left lower extremity had been for six months affected by a swelling which extended from the upper portion of the thigh down to the malleoli; there was also a slight arthropathy of the left knee. The femoro-tibial joint was enlarged, the patella raised by a small quantity of fluid, and the leg could be rotated and moved laterally much more extensively than in the normal state. The circumference of the left knee was four centimetres larger than that of the right. Cutaneous sensibility was hardly affected, while the muscular sense was much diminished, and ataxy was marked. Buzzard, who was the first to draw the attention of the profession in this country to the arthropathy of tabes, believes, from the frequent co-existence of gastric crises with that condition, that a lesion of a structure adjacent to the nuclei of the vagus in the medulla oblongata may be found to explain the osseous affection; but as neuritis and consequent atrophy of articular nerves has now been shown to exist in such cases, this theory, which presupposes the existence of an unknown and problematical centre for the nutrition of joints and bones, will have to be definitively abandoned.

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[1] "Archives de Physiologie," p. 160, etc. Paris, 1868.

CHAPTER III.

PATHOGENESIS OF TABES.

HAVING in the last chapter given a concise description of the morbid appearances in the different portions of the nervous system met with in tabes, we have now to consider the question which of the numerous alterations that we have found to exist is the primary and essential one, and what is the exact pathological nature of the change. It is quite impossible for me to even allude to all the numerous theories which have from time to time been started with reference to these points, and I must confine myself to the discussion of the newest and most important amongst them.

1. When Duchenne was under the impression that he had discovered a new disease, "locomotor ataxy," he thought that the *cerebellum* must be the seat of the malady; and more recently Neftel, of New York, has asserted that tabes is really an affection of the *brain*, and that all other alterations which may be found must be secondary to the cerebral lesion. But what does pathology teach us? We have seen in the preceding chapter that, as a rule, the morbid alteration stops short in the medulla oblongata, and that changes in the cortex and other portions of the brain have only been found in exceptional instances, where towards the end tabes had become complicated with epilepsy, general paralysis of the insane, and other similar conditions. It is quite true that we meet occasionally in the initial stage of tabes with temporary mental affections, and with aphasia, apoplexy, hemiplegia, and other

symptoms pointing to cerebral disturbance; but all these symptoms are fleeting and evanescent. They occur chiefly, and perhaps exclusively, in syphilised subjects, who are, some years after the primary affection, liable to similar symptoms, even where no signs of tabes have made their appearance; and they are part and parcel of the congestive form of brain-syphilis. They are also liable to occur in alcoholism, senile dementia, general paralysis of the insane, and insular sclerosis. There is loss of consciousness, aphasia, monoplegia or hemiplegia, coming on suddenly and lasting an hour or two, or at most a few days, or a week, showing that there is no serious brain-lesion.

The theory of the cerebral origin of the disease seems, therefore, to rest only on certain somewhat imperfectly understood clinical observations; while the pathological evidence of innumerable well-observed cases is directly antagonistic to it; and I therefore do not consider it necessary to adduce further arguments against its acceptance.

2. Another theory, which is only held by a few, is that that universal scapegoat, the *sympathetic system of nerves*, is at the bottom of the complaint. We have seen that disintegration of the fibres and ganglionic cells of this portion of the nervous system does undoubtedly occasionally occur in advanced cases of tabes. But to conclude from this that the sympathetic is primarily affected, and and that thence springs alteration of blood-vessels and wasting of nerve-tubes, constitutes a *salto mortale* which is somewhat too hazardous for us to follow. No doubt the principal reason for rejecting this view is that the alterations alluded to are exceptional. Moreover Vulpian has very properly drawn attention to the circumstance that, even in health, the more intimate structure of the sympathetic is by no means invariably the same. Great variations occur in healthy subjects in the proportion of nerve-fibres furnished with a medullary sheath and

Remak's fibres, and also in the size and pigmentation of the ganglionic cells. But even supposing that there was a constant and well-demonstrated change in the sympathetic nerve, it would even then be impossible to explain why this should lead as a rule only to affection of the posterior columns, and not of the other constituents of the spinal cord.

3. Tacász and others have broached the view that the primary event is atrophy of the *posterior nerve-roots*, and that the disease creeps from there upwards to the substance of the cord itself, like an ascending neuritis. It is quite true that at an advanced stage of the malady the posterior roots are habitually as much affected as the posterior columns ; but the roots never suffer to the exclusion of the columns. Moreover, such careful observers as Jädersholm, Westphal, and Tuczek, have, in early cases of tabes, found the roots healthy when there was already unmistakable disease in the columns; and this is a death-blow to the posterior-root theory.

4. A theory recently brought forward by Herbert Page[1] seeks the starting-point of this formidable malady, at least sometimes, in *a corn!* Here we seem to have arrived at the infinitely little, in order to account for a great deal. The prevention of tabes would, according to this theory, rest with the chiropodists. Page says that tabes dorsalis may in some cases have a peripheral beginning, and that a painful corn under the metatarso-phalangeal joint is a by no means trifling affection, little deserving of treatment. Perforating ulcer is, according to him, a symptom or consequence of some nutritive derangement of the affected part, and in such cases the disease may have really begun at the peripheral parts of the nervous system, so that the nerve-lesion was originally caused by the continued painful—often severely painful—

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[1] "Brain," October, 1883, p. 368.

pressure of the corns, the protracted sensory disturbance giving rise to ultimate structural change ; and that in the course of time the degeneration, travelling upwards, reached the spinal cord, and then, and only then, gave rise to the symptoms of tabes dorsalis ! This author thinks that his view is supported by the recent researches of Pitres and Vaillard on non-traumatic peripheral neuritis, and adds an instructive case, in which a corn seemed the starting-point of all the troubles which the patient subsequently experienced.

It appears to us that the discomfort which the majority of Europeans habitually experience from corns is bad enough, without crediting these little pests with such wickedness as eventually to conduce to tabes. That corns, especially in certain positions, may be often the beginning of nutritive changes in a limb seems probable enough. But in the case just mentioned an important link is wanting, inasmuch as it has not been shown that the patient was free from disease of the cord at the time when the corn commenced to give trouble. The sequence of events is much more likely to be as follows :—Tabes, from whatever cause, is the primary change. This induces liability to peripheral nerve-disturbance, so that certain exciting causes, such as long-continued pressure or injury to structures, are more likely to lead to mischief than in healthy persons whose vitality is not defective, and in whom the nutrition is not impaired by disease or degeneration of the nervous centres. In this respect the observations of Pitres and Vaillard are highly suggestive, as showing that the peripheral nerve-lesion is not by any means continuous between the distal extremity of the nerve and the spinal cord, but that, on the contrary, the large nerve-trunks between the periphery and the cord are healthy. There is, therefore, no creeping upwards of a peripheral neuritis to the cord, but we have rather to do with neuritis excited by a local cause and confined to peripheral nerve-terminations, but promoted by insufficient resistance in the nerve-centre previously diseased. Moreover, it has been shown that this form of peripheral neuritis is not by any means confined to tabid subjects, but that it is also liable to occur after cerebral hæmorrhage, fracture of the skull, herpes zoster, leucocythæmia, alcoholism, smallpox, diphtheria, and other distempers.

5. The changes which I have described as habitually found in the *pia mater* (p. 14) have induced some observers, such as Arndt, Waldmann, and others, to think that leptomeningitis is the primary event, and the actual cause of the wasting of the posterior columns, and that the change in the cord is a secondary degeneration. This opinion, however, is contradicted by two series of facts, viz., first by cases of undoubted meningitis, in which the lesions peculiar to tabes have been absent; and, second, by cases of initial tabes, in which no spinal meningitis existed. As tabes has in general no tendency to shorten life, cases in the first stage of the disease rarely come on the post-mortem table. Quite recently, however, Strümpell has carefully examined the cord of a woman who had only for a short time suffered from the malady, and who had died of typhoid fever. She had had lightning pains for two years, and showed absence of knee-jerk, reflectory pupillary rigidity, and ptosis of the left upper eyelid. To the naked eye the cord appeared healthy; but on microscopic examination it was discovered that the posterior columns were affected with degeneration in their entire extent, from the middle portion of the cervical region down to the lower portion of the lumbar region. Yet there was no spinal meningitis. Again, Tuczek has lately examined four cases of initial tabes, which had been produced by ergotism, and found sclerosis of Burdach's columns from the lumbar portion up to the medulla oblongata; yet in all of them the pia spinalis was perfectly healthy. It has also been occasionally noticed that there was no exact proportion in the degree

of morbid changes found in the membrane, on the one hand, and in the cord on the other hand.

As the distribution of blood-vessels is the same throughout the different portions of the pia spinalis, showing no peculiarities whatever in the part corresponding to the posterior columns, which would distinguish it from that investing the antero-lateral columns, it has been considered singular that the inflammatory appearances should be quite confined to the posterior portion of the membrane. It has been argued that this is owing to the exquisite sensibility which exists in the corresponding portion of the cord, where any irritative influence is more likely to be felt and resented than in the antero-lateral columns. These latter have, it is true, a slight degree of sensibility, but it is recurrent, not very keen, and certainly not to be compared with that of the posterior columns. When an irritant influence, therefore, acts on the superficial portion of the posterior columns, it would *primâ facie* be probable that it would also act on the corresponding portion of the pia. It is in the posterior portion of the spinal pia that we see the characteristic granulations of subacute tubercular meningitis ; and the localisation of these can certainly not be considered accidental. These considerations, however plausible, seem yet controverted by the circumstance that posterior lepto-meningitis is not constant, and occurs only in the later stages of tabes, when not only finer changes have taken place in the intimate structure of the nerve-tubes, but when gross alterations are perceptible to the naked eye, and the size and consistency of the posterior columns have been markedly affected. Perhaps it will appear to be, therefore, more in accordance with all the facts known at present to assume that the process is rather mechanical in character, and that the thickening of the pia helps to fill up the vacuum which is produced by the shrinking and wasting of the posterior columns. The view that the peculiar lightning pains of tabes are caused by inflammation of the pia can certainly not be maintained, as these have been observed long antecedent to any alteration of that membrane.

6. All our present real knowledge of the pathology of tabes converges, therefore, to the conclusion that the disease is primarily one of the spinal cord, and more especially of its *posterior columns.* Differences of opinion, however, still exist concerning the tissue in which the malady actually begins. Is the fault primarily located in the blood-vessels, the neuroglia, or the nerve-tubes themselves?

a. The *blood-vessels* of the cord have, by Ordoñez, been said to be the starting-point of the degeneration. This able histologist has described an initial lesion of the arterioles of the posterior columns, the coats of which he found choked up with oil-globules and granular corpuscles. Such a condition, according to him, must needs conduce to imperfect exchange of nutritive material between the vessels and the tissues, and therefore to atrophy and sclerosis of the nervous structures. This view of Ordoñez, which has been broached with considerable acumen, has, however, been controverted by Vulpian,[1] who objects to it that, while the lesions of the nerve-tubes in tabes are constant, the vascular changes are variable and often absent. Moreover, it should be remembered that alterations in the blood-vessels similar to those occasionally observed in tabes occur in Wallerian degeneration, that is, after experimental section of nerves in animals; and that they are for this reason more probably the consequence than the cause of the changes which take place in the nervous structures.

The views of Ordoñez have, however, quite recently found a striking confirmation by Bevan Lewis,[2] who found in a cord sent to him by Buzzard for examination, most unquestionable appearances of peri-arteritis in the blood-vessels of the posterior columns, which had apparently originated in the membranes, more especially the posterior portion of the pia, and had from there spread inward along the vessels of the posterior columns. The vessels of the latter were extremely numerous and dilated ; their coats thickened and diseased. The wide-stretched orifices of these numerous vessels formed invariably the centre of a patch of fasciculated sclerosis. In vertical sections of the sclerosed parts diseased vessels were seen in large numbers with ampullar enlargements along their course, which were owing to vast accumulations of large nucleated cells occupying the peri-vascular sheath of the vessel. The actual calibre of the vessel was sometimes dilated ; at other times it was constricted by the encircling mass of cells ; whilst occasionally, although rarely, a genuine aneurismal condition of the vessel had been produced along its diseased tunics. It therefore appeared that the whole dorsal region of the cord had been subjected to an invasion by an extensive peri-arterial affection, and its medullated strands exposed to destructive pressure from nodular outgrowth on the vessels. The peri-arteritis, originating in the membranes and spreading inwards to the posterior columns, would therefore, in that case, have played the chief part in the production of the lesion to which both Burdach's and Goll's columns had succumbed.

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[1] "Maladies du Système Nerveux," p. 385. Paris, 1879.

[2] "Brain," p. 467. January, 1884.

Bevan Lewis's account of the post-mortem appearances in this case is a model of what such descriptions should be, and can leave no doubt whatever on the mind that cases occur in which the vascular change is the primary one ; yet probably such cases are rare, as many good observers appear never to have met with them.

b. Another theory which has been brought forward more particularly by Adamkiewicz is that the essential lesion of tabes consists of interstitial degeneration of intertubular connective tissue, and not of primary degeneration of nerve-fibres. According to this author, the connective tissue becomes sclerosed in tracts corresponding to the course of the blood-vessels, and the sclerosis, once commenced, has the tendency to creep on in the interstices of the parenchyma of the cord. Wherever connective tissue enters from without into the posterior columns, that is, at the posterior fissure of the cord and at the boundary between Burdach's and Goll's columns, there the sclerosis is active, and strangles the nerve-fibres. This he believes to be in consonance with the theory of the syphilitic origin of tabes, as syphilis is known to give rise to interstitial degeneration; and the process is said to be analogous to cirrhosis of the liver, which consists likewise of interstitial degeneration of connective tissue.

This view, which has been very confidently put forward, is based on the observation of a very small number of cases, none of which appear sufficiently well marked to carry conviction to our mind, even as far as they go. On the other hand, a much larger class of cases is on record which speak directly against the hypothesis just mentioned; and what is even more important is the circumstance that, if we were to accept this theory we should find it impossible to understand why a disease which primarily affected the neuroglia should be so strictly localised to certain systems of the cord as tabes has been shown to be. The anatomical distribution of the connective tissue is, like that of the blood-vessels, exactly the same in all the different systems and strands of the cord; whereas we have seen that the nerve-tubes form separate and perfectly distinct groups and systems, which belong evolutionally together, and become successively developed at special periods of embryonic life, constituting functionally separate groups, of which it is easy to understand why they should respond differently to various injurious influences which may be brought to bear upon them during some period of the struggle for existence.

Another reason which speaks against the connective-tissue theory is, that we find, in cases where there is unquestionably interstitial inflammation of peripheral nerves, as in some forms of neuritis descendens, plenty of healthy nerve-tubes left, while the neurilemma and the interstitial connective tissue are considerably proliferated. If therefore the neuroglia were primarily affected in tabes, we should no doubt discover numbers of healthy nerve-tubes in the posterior roots and columns, together with overgrowth of the glia. Such, however, is not the case, and this also leads to the conclusion that in tabes parenchymatous disease precedes interstitial disease.

c. While, therefore, general considerations, based on well-ascertained evolutional and functional differences between the several tracts of fibres in the cord, point clearly to the conclusion that the *central nerve-tube* is primarily affected in tabes; and while direct observation of glia-changes has failed to prove that they arise independently of tube-changes, we have, nevertheless, in the last resort to appeal to direct observation of the alterations found in the nerve-fibres for the decision of this question.

Now it is found that the first pathological change which takes place generally affects the axis-cylinder of the central nerve-fibre. From a functional point of view, this is the principal constituent of the nerve-tube; and most probably irritation of it gives rise to those attacks of lightning pains, which form in general the most prominent symptom of the first stage of the disease. Repeated attacks of irritation lead to imperfect nutrition, and finally to atrophy. The dead axis-cylinder then acts as a foreign body, and causes further irritation in the neighbourhood. The nuclei of the nerve-fibres swell and multiply; the protoplasma surrounding the nuclei begins to proliferate, the myeline sheath becomes segmented, and ultimately reduced to small drops and granulations, which eventually disappear in their turn. The irritation is now likewise propagated to the intertubular connective tissue, the cells of which begin to multiply, while the fibres proliferate. The coats of the blood-vessels then participate in the disease, and the white blood-globules are changed into a granular mass. Such a morbid process therefore appears to be rather one intermediate between atrophy and inflammation; and this, apart from everything else, fits in much better with other facts, inasmuch as we can readily understand why atrophy should be limited to certain sets of fibres; while, if inflammation invaded the structures, one does not see why it should not spread contiguously to other tracts of nerve-tubes, like ordinary myelitis, seeing that the distribution of the connective tissue and of the blood-vessels is everywhere uninterruptedly the same.

On reviewing the whole of the morbid changes which are found to take place in tabes, they appear to us of an extremely complex character. They certainly do not constitute simply "sclerosis of the posterior columns," although this is undoubtedly the most important and fundamental lesion. Nor can we quite agree with the ingenious and brilliant theory of the disease which has been put forward by Pierret.[1] According to this observer, tabes consists anatomically of inflammatory alterations which have their principal seat at two points, viz., 1st, in the peripheral receptive organs, such as the retina, the auditory nerve, etc., and 2nd, in the columns and sensory ganglia of primary and secondary reflex action.

The principal symptoms therefore consist of various disturbances of general and special sensibility, while the disturbances of motor function, which likewise occur, are to be explained by the intimate relations existing between the motor and sensory tracts; and those disturbances which occur in the vascular sphere, must be referred to implication of the vaso-motor system, which is intermediate between the motor and sensory tracts, passing from the cord to the medulla oblongata, through the medium of the posterior pyramids. The disease is therefore by Pierret and his pupils often called " sensitive tabes."

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[1] "Essai sur les Symptômes céphaliques du Tabes Dorsalis," Paris, 1876; and "Transactions of the International Medical Congress of London," 1881, vol. i., p. 399.

Now there can be no doubt that " all movements are performed under the influence of sensory impressions, and that the motor nerve nuclei are educated for co-ordinated action by corresponding sensory nuclei." (Broadbent.) This theory will explain the phenomena of ataxy, but is insufficient to account for the occurrence of actual paralysis. It is, however, a fact that, to mention only the cranial nerves, the third and sixth, which are purely motor in their function, are frequently affected in tabes, while paralysis of the portio dura and the motor portion of the fifth nerve has likewise been observed. In order, apparently, to get over this difficulty Pierret states that in certain fishes, such as Lepidosiren, the muscles of the eye receive their branches from the fifth nerve, and that in the Amphibia the sixth nerve, which supplies the rectus externus muscle, is not separated from the fifth. These considerations, which are derived from compared anatomy, are, however, not applicable to human physiology or pathology; nor is it possible to agree with Pierret in thinking that all palsies which occur in the course of tabes have to be looked upon as reflex neuroses.

Paralysis of the rectus externus occurs sometimes years before the appearance of lightning pains or other sensory disturbances, and cannot by any stretch of imagination be looked upon as reflex paralysis. Then, again, paralysis of certain sets of muscles of the body and limbs is not at all uncommon in tabes ; and so is muscular atrophy, owing to wasting of the ganglionic cells in the anterior cornua of the grey matter. All these signs appear to us totally incompatible with the sensory theory of tabes, which, although applicable to a considerable number of the symptoms which are observed in that extraordinary disease, cannot cover the whole of them, unless some obstinate and unruly ones are placed in the bed of Procrustes, to be either cut off altogether or to be pulled out of all their original shapes, so as no longer to resemble their natural selves. The time has evidently not come yet for "crowning the edifice," that is, for building up a theory of tabes which will account for all phenomena of the malady; and a great deal of painstaking observation will be required before this desirable object has any chance of being accomplished. *What we can say with certainty is that the anatomical as well as clinical features of tabes are of an extremely complex character; that they vary greatly in different cases; that the mode in which they are grouped together has, as yet, not been found to follow a definite law; and that it would be premature to commit us to any positive or dogmatic view of the nature of the disease.*

CHAPTER IV.

MORBID ANATOMY OF OTHER FORMS OF SCLEROSIS.

1. *Primary sclerosis of the lateral columns.*—The next form of sclerosis which we have to consider is that which is believed to form the material base of *spastic spinal paralysis,* or *spasmodic tabes dorsalis,* and to consist of *primary symmetrical sclerosis of the lateral columns.* This disease, the symptoms of which are well known, seems as yet to have eluded the grasp of the pathologist. Ross,[1] of Manchester, attributes to his colleagues, Professors Morgan and Dreschfeld, of that city, the honour of having been the first to prove by dissection the connexion of the symptoms of spastic paralysis with lateral sclerosis in a primary and uncomplicated case of the disease, and says that they found symmetrical sclerosis of the crossed pyramidal strands from the medulla oblongata to the conus medullaris, *and an entire absence of any other lesion.* The same opinion is expressed by Charlton Bastian[2] and by Russell,[3] of Birmingham. The account, however, given by Dreschfeld, both in the "Transactions of the International Medical Congress," and in the "Journal of Anatomy" (1881), shows quite clearly that his case was not one of uncomplicated lateral sclerosis, *inasmuch as the giant-cells of the grey anterior cornua were likewise affected.* He says

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[1] "A Treatise on Diseases of the Nervous System," 2nd edition, vol. ii., p. 82. London, 1883.

[2] "A Dictionary of Medicine," by Richard Quain, M.D., F.R.S., p. 1486. London, 1883.

[3] "Medical Times and Gazette," Feb. 11, 1884.

that in his case the brain and medulla oblongata were apparently healthy; that the spinal cord showed to the naked eye nothing but slight softening in the dorsal region; but that the microscopic examination of the specimen showed the neuroglia in the anterior pyramids of the bulb to be slightly increased, the anterior and lateral pyramidal strands extensively diseased, chiefly in the cervical and lumbar region of the cord; while the multipolar ganglionic cells of the anterior grey cornua were found wasted from the upper dorsal region down into the lumbar portion of the cord. There was simple atrophy, pigmentary atrophy, and occasionally complete absence of whole groups of these cells. The case is therefore seen to be one of the amyotrophic variety of the disease, and not one of uncomplicated lateral sclerosis.

Most other cases which have until now been recorded are equally unsatisfactory, for one reason or another. Charcot and Pitres have described one which was during life diagnosed as one of primary lateral sclerosis, but turned out on the post-mortem table to be one of insular disseminated sclerosis. Schultze has described four cases in which the symptoms of lateral sclerosis were present; and eventually discovered in two of them a tumour of the brain, while the third was one of chronic internal hydrocephalus, the latter even without a trace of sclerosis. In another case there was found myelitis from compression, or rather dorsal hypertrophic pachymeningitis, the dura mater being very much thickened, more particularly laterally, and showing purulent detritus on its inner surface, the pia having become attached to it, and the cord being flattened, compressed, and softened. Aufrecht and Hopkins have more recently recorded cases in which there were the symptoms of spastic paralysis during life, and lateral sclerosis was discovered after death, again, however, complicated with atrophy of the anterior cornual cells. In Hopkins's case the lateral columns and the adjoining portion of the anterior columns proved to be nearly devoid of nerve-tubes at the lower end of the cord; while in the anterior columns overgrowth of intertubular connective tissue was noticed. Higher up in the cord more tubes were seen, and in the mid-dorsal region the anterior columns were nearly normal. Hopkins does not state whether the sclerosis was confined to the pyramidal tracts, or whether it also affected the direct cerebellar column; but the large cells of the anterior cornua were much atrophied in the lower half of the organ, except in a portion taken from the lumbar enlargement; while a little lower down, where the cells were arranged in three well-defined groups, viz., a central, a larger antero-lateral, and a large postero-lateral, the two first groups were markedly atrophic, more especially on the right side.

The only case which appears to me to be one of undoubted and uncomplicated primary lateral sclerosis is one which has quite recently been recorded by Minkowsky.[1]

This occurred in the University Hospital of Königsberg, under the care of Professor Naunyn. It was that of a girl, aged 19, who was admitted for secondary syphilis in May, 1881. The date of the primary infection could not be ascertained. She was treated by inunction, and got apparently well; but fresh specific eruptions having shortly afterwards supervened, she had to be re-admitted. Symptoms of phthisis presently came on, and likewise great weakness in the lower extremities, accompanied with tremor. In January, 1882, she could not walk without support; when supported, the gait was shuffling; she could hardly lift the toes from the ground, and there was much sclerotic tremor. The upper extremities appeared feeble, but not paralysed. Sensibility was everywhere normal. The cutaneous reflexes were absent, but the kneejerk was exaggerated on both sides, and there was ancle-clonus. There were no symptoms on the part of the bladder and rectum. Inunction was now again resorted to, and in a little more than a week the tendon reflexes appeared to be much less marked. Shortly afterwards the walk became more certain, and the tremor ceased. A month after admission the patient walked tolerably well without support. The phthisical symptoms, however, increased, and the general condition of the patient now became so bad that the treatment by inunction had to be discontinued. Hectic fever supervened, and the girl died in July, 1882. The inspection showed to the naked eye a slight reduction of the postero-lateral compared with the antero-lateral columns; otherwise no change was perceptible to the unassisted eye. After having been hardened in Müller's fluid, the postero-lateral columns showed a much lighter colour, more especially in the dorsal portion of the cord, and corresponding to the crossed pyramidal and direct cerebellar column. Sections of the dorsal cord showed under the microscope that the disease was limited to the strands just mentioned. In the crossed pyramidal columns the number of nerve-tubes was much diminished, but there were numerous fibres in a perfectly normal state. Some were thin, with myeline-sheath atrophied, and there were numerous lacunæ which evidently corresponded to wasted nerve-tubes, and where thin remains of axis-cylinders might still be recognised. The neuroglia was somewhat overgrown, and contained numerous nuclei, with dilated blood-vessels whose adventitia was thickened. There were a few oil-globules and amyloid bodies. In the direct cerebellar column the nerve-tubes appeared almost entirely perished; the intertubular substance was considerably proliferated, and there were no lacunæ. All the other strands of the white matter were perfectly normal. In the whole of the dorsal portion of the cord the *anterior cornua of the grey matter were perfectly normal;* the number of ganglionic cells was large. In Clarke's vesicular columns the cells appeared slightly diminished, and some of them contained a good deal of brownish pigment. There were, however, no certain signs of actual degeneration there.

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[1] "Deutsches Archiv für klin. Medicin," vol. xxxiv., p. 433. 1884.

In the *lumbar* cord the wasting of nerve-tubes in the affected strands was much less marked, and the grey matter perfectly healthy. In the *cervical* cord there appeared to be a good deal of difference between the crossed pyramidal and the direct cerebellar strands, inasmuch as the former showed very slight, and the latter a very marked, degeneration. Goll's columns appeared at first sight to participate somewhat in the disease, but on closer examination this was shown not to be the case. The grey matter was perfectly normal in the upper portion, while in the lower there was a slight effusion of blood at the boundary between the anterior and posterior horns.

This case is also interesting as showing that spastic paralysis may occur in consequence of syphilis. The patient was a girl without inherited neurotic tendency; the disease appeared after infection, and was nearly cured by specific treatment. It also shows that even when symptoms of spinal disease disappear, the lesion which originally caused them may still be present. Schultze has seen a case of tabes where the same occurred.

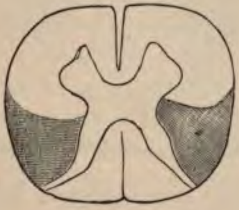

FIG. 7.

Primary lateral sclerosis.

According to Charcot and Bouchard, the pyramidal column is, in primary lateral sclerosis (Fig. 7), found studded with wedge-shaped grey patches, reaching anteriorly as far as the lateral column proper, exteriorly as far as the pia mater, and interiorly as far as the posterior cornua. This peculiarity in the localisation of the disease is said to distinguish it anatomically from Türck's degeneration, or *secondary lateral sclerosis* consequent on cerebral hæmorrhage or softening, in which the patches are rounded, not wedge-shaped, and do not spread exteriorly as far as the pia mater; and likewise from secondary sclerosis after myelitis, hæmorrhage into the cord, etc., (Fig. 9,) where the grey patches do not extend posteriorly as far as the posterior cornua, but where a small intermediate layer of healthy white substance remains between them and the sclerosed tissue. It has not yet been ascertained

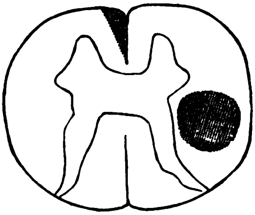

FIG. 8.

Secondary lateral sclerosis after cerebral disease. (Türck's degeneration.)

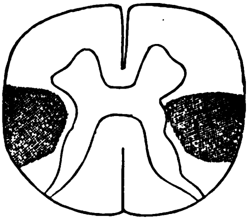

FIG. 9.

Secondary lateral sclerosis after myelitis.

whether these minute differences are at all constant in the different forms of lateral sclerosis, and the whole subject is still involved in much obscurity.

Leyden has long ago expressed his disbelief in Erb's spastic paralysis as a distinct disease; and has, in cases which showed the symptoms described by Erb during life, generally found myelitis of the dorsal portion of the cord. According to him these symptoms may be owing to myelitis from injury, in the later stages of which muscular spasms and rigidity are apt to make their appearance; to myelitis from compression of the cord by carious vertebræ or tumours; to syphilis; to meningitis, or pio-myelitis of acute or sub-acute course; and finally to that form of paralysis which follows certain acute diseases, such as typhoid fever, smallpox, relapsing and intermittent fever, and the puerperal state.

Apart, however, from these affections of the spinal cord and its membranes, the phenomena of spastic paralysis may be owing, particularly in children, to disease of, or deficiency in, the motor area of the cortex, or of the white conducting strands connecting the latter and the anterior cornua of the cord. This may be congenital, and a consequence of what Heschl has called *porencephaly*. If there is a porencephalous defect in the central convolutions of the brain, viz., the ascending frontal and the ascending parietal, then the downward passage of the pyramidal strands, which proceed from the ganglionic cells of the cortex through the crus, pons, and medulla oblongata to the spinal cord, must be arrested. If such arrest takes place high up, all four extremities would be affected; while if lower down, only the legs would suffer from spastic paralysis. In almost all such cases there would, however, be at the same time epileptiform fits, more or less imbecility, and cerebral symptoms. Meschede has described a case where there was a defect in the parietal bone, through which the brain could be seen to pulsate. After death there was found immediately beneath the defect in the bone, in the right hemisphere, a cavity three inches long and proportionately wide and deep, filled with serous fluid. This communicated with the lateral ventricles; and both ventricles were so much distended by fluid that the cortex as well as the basal ganglia were compressed. The pyramidal strands were undeveloped. Kundrat has recorded an analogous case, in which the left hemisphere near the island of Reil was the seat of a similar cavity; the lateral ventricles were distended by fluid, and the pyramidal tracts of the cord completely undeveloped. Similar cases have more recently been described by Ross and Hadden. Where delivery is tedious, more especially in the case of a first child, the brain may suffer injury, either by hæmorrhage, softening, or local encephalitis, leading to atrophy of the motor area of the cortex and sclerosis of the pyramidal strands. The symptoms of lateral sclerosis may also be complicated with general paralysis of the insane.

Secondary sclerosis of the pyramidal strands is, however, most frequently seen after attacks of cerebral hæmorrhage, involving the motor convolutions of the cortex of the brain, or the corpus striatum, and after embolism and thrombosis of the blood-vessels supplying these parts, more especially the left middle cerebral artery, leading to softening of the motor zone of the brain. The degree and extent of this sclerosis varies. In some cases the whole texture of the pyramidal strand has been found utterly destroyed, there being no nerve-tubes left, but only amyloid corpuscles and overgrown connective tissue; while in other cases the degenerative process appears to be more limited, there being white patches interspersed in the grey mass, containing healthy nerve-tubes. The extent of the primary lesion in the brain determines the degree of the secondary sclerosis in the pyramidal strand.

The sclerosed appearance of this tract of fibres begins immediately behind the lesion, and follows its anatomical distribution further down. It is particularly marked in the crus cerebri, more especially its middle portion. In the pons Varolii it is not so obvious, but becomes again more clearly apparent in the anterior pyramid of the medulla oblongata, where it crosses over to the opposite side of the cord, and runs down in the posterior portion of the lateral column until it reaches the lumbar enlargement. This sclerosis leads to irritation in the motor cells of the anterior horns of the spinal cord, and thereby causes rigidity of the paralysed muscles, which is an exaggeration of their normal tonicity. It is only in exceptional cases that the sclerosis spreads from the pyramidal strand to the anterior horns, and induces wasting in the giant cells of the grey matter. Destruction of the centre of muscular tonicity then leads to atrophy of the paralysed muscles, which at the same time lose their rigidity. All grey cells being connected, the process may spread from the anterior to the posterior horns, or to the anterior horns of the opposite side, by means of the anterior commissure; and we have then no hemiplegia, but paraplegia with muscular atrophy. Some of the ganglionic cells may be entirely gone, while those which remain look sickly, yellow, and granular, and seem deprived of their nuclei and Deiters's elongations. The intermedio-lateral cells, which are believed to be centres of vasomotor action, have likewise occasionally been found destroyed

In cases where decussation is incomplete, sclerosis is found in Türck's direct pyramidal column (P, Fig. 2, p. 7). Flechsig has shown that total crossing of the fibres from one to the other side only takes place in a certain number of cases; in others there may be total crossing of one pyramid and semi-decussation of the other, or there may be semi-decussation of both strands. In such cases more than fifty per cent. of the fibres of the pyramidal strand may remain uncrossed, and then proceed straight on to the anterior columns of the same side.

Sclerosis may therefore be discovered in both the crossed and uncrossed pyramidal strands. Bouchard states that the fibres of Türck's columns gradually vanish in the dorsal region of the cord ; and that in the lower portions of the organ, therefore, only the crossed pyramidal column is found sclerosed. Similarly, in compression of the cord, descending degeneration is said to be found in Türck's columns only, if the seat of the lesion is high up. This statement has been repeated by most subsequent writers on the subject, but seems really to apply only to a limited number of cases.

Sclerosis of that portion of the lateral column which is known as the direct cerebellar strands is generally connected with other forms of sclerosis, more particularly of Goll's and the pyramidal strands columns, and seems to be always secondary and ascending. It occurs in Pott's disease, and is generally associated with wasting of Clarke's vesicular columns, which are considered to be the trophic centre of the direct cerebellar strands.

2. *Sclerosis of Goll's columns.*—Primary sclerosis of Goll's or the postero-internal columns apart from any other lesion is excessively rare, as up to the present time only three such cases have been placed on record, viz., by Pierret, Du Castel, and Gowers. Combined and secondary sclerosis of these columns is, however, frequently discovered. It is habitually found in cases of Pott's disease in the dorsal region of the cord, and other affections of the white medullary matter lower down. It is then called ascending degeneration, and is, as I have just mentioned, apt to occur together with sclerosis of the direct cerebellar strands. It does not follow disease of the grey matter of the cord, such as atrophy of the ganglionic cells of the anterior horns, in infantile paralysis and progressive muscular atrophy. The crossed pyramidal strands may be found sclerosed together with Goll's columns and the direct cerebellar strands. All these strands contain long fibres, and are

evolutionally distinguished by a late formation of their myeline sheaths.

Ascending sclerosis of Goll's columns has been experimentally produced by Singer, who has seen it to occur after section of the posterior roots of the sacral and lumbar nerves; and from this it is concluded that these columns consist almost exclusively of fibres which ascend from the roots just mentioned directly towards the medulla oblongata. An experiment made by Kahler leads to the same conclusion. He injected liquid bees-wax into the epidural space from the sixth dorsal vertebra, and this when solidified compressed the cord as well as the nerve-roots. The invariable consequence was degeneration of the upper dorsal and lower cervical roots, and as a secondary consequence ascending degeneration of Goll's columns.

3. *Amyotrophic lateral Sclerosis.*—This is a peculiar form of sclerosis, which was first described by Charcot, and is distinguished by simultaneous affection of the lateral columns of the cord and of the large ganglionic cells of the anterior grey cornua. It seems almost invariably to begin in the cervical region of the cord, and has the tendency to spread from there both upwards and downwards. The dorsal and lumbar portions are, therefore, gradually affected in the same manner, and the disease eventually creeps upwards towards the medulla oblongata, where the motor nuclei are found sclerosed. Charcot's observations have been amply confirmed by other pathologists.

In a case which has recently been very carefully described by Stadelmann, the crossed pyramidal columns showed in the cervical cord numberless oil-globules, fewer nerve-fibres than usual, and overgrowth of neuroglia. The direct cerebellar columns and Türck's columns were healthy. In the pyramidal tracts there was the peculiar "pin-hole" appearance corresponding to perished nerve-fibres, the vacuum being filled up by a pale, finely granular substance. In the anterior cornua was at the same time found great diminution of the giant-cells, more especially in the median and antero-lateral group, while in the intermedio-lateral tract they appeared undiminished. The cells which still existed in the anterior cornua were either shrunk or swollen, pigmented, and had only short processes. The anterior roots were thin, and looked wasted; while Clarke's vesicular columns were normal.

In the dorsal cord the disease of the crossed pyramidal strands was much more severe. There were very few fibres left; the oil-globules and pin-holes were abundant; and the degeneration extended posteriorly to the posterior columns; while also the antero-lateral zone and the whole of Türck's column was diseased. The anterior cornua were more affected than in the cervical cord, while in the lumbar cord the affection was much less. The pyramidal strands appeared wasted in the medulla oblongata; the grey matter on the floor of the fourth ventricle, including the nuclei of the hypoglossus and spinal accessory nerve, was atrophied; any ganglionic cells which still existed appeared either shrunk or swollen, pigmented, and almost devoid of protoplasmic or nerve-processes.

4. *Insular or disseminated Sclerosis* (Sclerosis in patches).—There are three forms of this disease, viz., the *spinal*, which affects different areas of the spinal cord; the *cerebral*, which invades chiefly the motor zone of the brain; and the *cerebro-spinal*, in which the two former affections occur together. The merit of having first distinguished this form of sclerosis belongs to Vulpian, and more particularly to Charcot and his pupils.

In insular sclerosis, multiplication of nuclei and proliferation of the fibres of the neuroglia constitute the initial fact, while degenerative atrophy of the nerve-tubes is consecutive and secondary.

In spinal insular sclerosis the patches are chiefly found in the antero-lateral columns, but they may exist indiscriminately in all the different portions of the cord, whether white or grey. The patches vary in size and shape from that of a millet-seed to a pea or small bean, and form a striking contrast with the healthy tissues in which they are imbedded. They have the same grey or yellowish coloration which is seen in the long bands of sclerosis with which we have become familiar as affecting entire strands or columns. Where there is much proliferation of connective tissue, these patches may be slightly elevated over the cut surface, while when there has been much shrinking there is a corresponding depression.

As far as their microscopical characters are concerned, Charcot distinguishes three different zones in these patches, viz., 1st, a *peripheral* zone, in which there is overgrowth of connective tissue and wasting of the myeline sheath, while the axis-cylinder is either normal or hypertrophied; 2nd, a *transition* zone, in which the medullary sheath has disappeared, the nerve-tubes have become attenuated, and the axis-cylinder enlarged; and, 3rd, a *central* zone, in which the neuroglia, with the myeline, has disappeared, and the axis-cylinder is reduced in size. A certain number of axis-cylinders, however, always persist in the otherwise completely altered tissue, which distinguishes this form of sclerosis from all others. The coats of the blood-vessels are thickened; amyloid bodies are found interspersed in the fibrillary tissue; oil-globules, which constitute the *débris* of the destroyed nerve-tubes, are plentiful in the peripheral portions, but absent in the central zone, where the morbid process is finished.

Klein, of Moscow, who has more recently investigated the anatomy of this disease, finds the earliest changes in the blood-vessels, which are dilated; the perivascular spaces contain lymphoid cells, the nuclei of the neuroglia are increased, and the bulk of the intertubular substance appears augmented. Later on the impaired circulation in the altered blood-vessels and undue pressure by the new cellular elements lead to degenerative atrophy of the nerve-tubes and nerve-cells. Finally, the neuroglia is replaced by sclerosed connective tissue, with occasional complete occlusion of the blood-vessels, whereby the nutrition of the diseased parts is still further affected.

It would, therefore, appear that at least in some forms of insular sclerosis there is an analogous change in the blood-vessels of the affected parts as that which has been described by Ordoñez and Bevan Lewis as occurring in certain forms of tabes. This seems also to be the opinion of Bastian,[1] who has found the walls of the capillaries, arteries, and veins greatly thickened, more especially the adventitia, and has seen this overgrowth to extend inwards so as to cause fibroid degeneration of the middle and inner coat of the vessel (endo-arteritis). Without committing himself to a definite opinion whether the process commences in the neuroglia or the blood-vessels, Bastian agrees with other observers in thinking that in this form of sclerosis the changes in the neuroglia are primary, and those in the nerves secondary; while in the bands of secondary degeneration the nerves suffer primarily, and the neuroglia subsequently.

The patches of primary insular sclerosis do not appear to conduce to any secondary degenerations, either ascending in Goll's columns, or descending in the antero-lateral columns. Most probably the reason of this lies in the peculiar anatomical character of the lesion, as, through the persistence of the axis-cylinder, the nervous influence is not entirely cut off between the fibres and their trophic centres. We should therefore expect to find any secondary degeneration only in very far-advanced cases of insular sclerosis.

5. Of other *combined system-diseases* of the spinal cord our knowledge is as yet very rudimentary. Kahler and Pick have described cases where the pyramidal strands, the direct cerebellar strands, and Goll's columns were

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[1] *Loc. cit.*, p. 1490.

diseased; and Strümpell has recorded some in which the patients showed during life the symptoms of amyotrophic lateral sclerosis, and in whom there were found after death combined degeneration of the pyramidal strands, a portion of the posterior columns, the direct cerebellar strands, and atrophy of cells in Clarke's vesicular columns. The white strands just mentioned are evolutionally distinguished by late formation of the myeline sheaths. In this affection there is also no transverse spreading of the disease, and each system is independently affected, as shown by different degrees of intensity of the affection in each, while the symmetrical character is fully sustained. As regards the posterior column, it appears that just those parts are affected which escape in the earlier stages of tabes, viz., the body of Goll's columns, the small round field at the anterior end of the anterior area, and the postero-external field (2, 3, and 5 in Fig. 3) in Burdach's columns.

Friedreich[1] in 1863 drew attention to a peculiar form of the disease to which he and others have applied the term *hereditary ataxy*. This name, however, appears to be an unsuitable one, as it seems to imply, first, that the disease usually known as tabes or ataxy is not hereditary; and, second, that the disease which Friedreich described, both clinically and pathologically, is identical with Romberg's tabes dorsalis or Duchenne's *ataxie locomotrice*. Such, however, is not the case, as hereditary influence is not altogether wanting in ordinary tabes, and the malady first described by Friedreich differs in so many essential points from ordinary tabes that we now consider the two affections to be quite distinct.

The cases originally recorded by Friedreich, were not such of direct transmission of a peculiar form of spinal disease from parents to children, but instances of a malady occurring in brothers and sisters whose parents had been free from it. The term "hereditary tabes" is therefore a misnomer. "Family ataxy," although more correct, does not sound well; and under these circumstances we may provisionally call the malady in question *Friedreich's disease,* which has the advantage of not committing us to any special pathological theory.

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[1] Virchow's "Archiv," vol. xxvi. and xxvii., 1863; vol. lxviii. and lxx., 1877 and 1878.

The pathological lesions in Friedreich's disease show a greater variety of degenerative changes than those which are habitually discovered in ordinary tabes. There is not only posterior lepto-meningitis and sclerosis in Goll's and Burdach's columns; but the lateral columns, Clarke's vesicular columns, the central grey matter, and even the anterior columns are found sclerosed. In the medulla oblongata the sclerosis extends to the posterior pyramids and the floor of the fourth ventricle, involving the nuclei and trunks of the hypoglossal nerves. The posterior roots have generally been found wasted and indurated, and the brachial, crural, and sciatic nerves in a state of atrophy.

Friedreich's disease, therefore, appears to be a diffuse sclerosis of different portions of the spinal cord and medulla oblongata, and clinically as well as pathologically distinct from tabes and from insular sclerosis. It is hardly worth while to discuss seriously Hammond's opinion, that the disease begins in the bulb, and afterwards invades the cerebellum; for we shall see further on that clinical observation shows it to commence in the lumbar enlargement of the cord, while pathological anatomy has shown the cerebellum to be healthy even after the affection had lasted upwards of thirty years. The complete collapse of Duchenne's first theory of locomotor ataxy being a cerebellar disease might have prevented Hammond from once more bringing the cerebellum forward in connection with the pathology of a similar complaint.

6. Finally, it has been shown that there is such a thing as *pseudo-sclerosis.* There may be clinically all the symp-

toms of sclerosis of the lateral columns, or of insular sclerosis, yet no lesion is discovered after death. In hysterical girls, Müller of Gratz, and my colleague, Hughes Bennett, have drawn attention to this occurrence of pseudo-lateral sclerosis, and Westphal has recently seen two cases of pseudo-insular sclerosis without anatomical lesion: one of these patients was a lad, who began to show signs of motor debility in the upper and lower extremities, and double vision at the age of eighteen ; six years later he became imbecile ; there was tremor in both upper and lower extremities attending any voluntary movements, and the phenomenon known as paradoxical contraction ; tremor was also seen in the head, tongue, and lower jaw ; the speech was drawling, and all movements were slow and clumsy. Three years afterwards the patient died, and all parts of the nervous system were found to be healthy. Some time ago I had a girl under my care at the hospital, who had been bedridden for three years previous to her admission, and who on examination showed all the symptoms of primary lateral sclerosis. Three months afterwards she left the hospital nearly well, and it therefore became quite evident that no structural lesion had existed, but that the case had been one of pseudo-lateral sclerosis.

CHAPTER V.

ETIOLOGY.

THAT certain poisons, when habitually taken into the system, may lead to sclerosis of certain portions of the spinal cord, cannot be disputed. It has long been known that a peculiar form of spinal debility is caused by eating bread mixed in undue proportions with ergot of rye, the permanent mycelium or sclerotium of a fungus of the family of the pyrenomycetes, which grows on the flower and fruit of secale cereale, and which contains, according to the researches of Dragendorff and Podwissotzki, two active principles, viz., sclerotinic acid (about 4·5 per cent.) and a colloid substance termed sclero-mucine (from 2 to 3 per cent.). The term ergotine, which was first introduced by Wiggers, and afterwards used by Bonjean and Wenzell, had better be avoided, as very different substances are comprehended by it; and it is by no means proved that what is called ergotine really constitutes the active principle of ergot. Even less is known of other alkaloids said to be contained in the mycelium, viz., ergotinine and ecboline. Sclerotinic acid, however, is a definite poison, five grains of which will kill a kitten, while ten grains prove fatal to a rabbit. The phenomena produced by the administration of this substance are, according to Nikitin, paralysis, lowering of the temperature of the body, and retarded respiration, which latter ceases before the heart's action is arrested. Tuczek has endeavoured to produce tabes artificially by feeding animals with small doses of ergot and sclerotinic acid. Under the influence of ergot, mice and fowls died rapidly, with symptoms of wasting and fatty degeneration of the principal organs. Rabbits, on the other hand, appeared to be insusceptible to the action of the poison, since they remained perfectly well when fed for months with doses varying from half an ounce to an ounce. Cats and dogs, again, wasted away, became unsteady, more especially in the hind legs; but the knee-jerk persisted, and after death no change in the spinal cord was discovered. Sclerotinic acid in small doses produced, according to the same observer, more distinctly the phenomena of locomotor ataxy, first in the hind-legs and afterwards in the fore-legs; but the knee-jerk could be elicited to the last, and inspection of the cord showed that organ to be unaffected. Experiments made with trimethylamin have likewise given uncertain data.

While, therefore, the results of purely experimental pathology are ambiguous, and so far bear out Vulpian's statement made some years ago, that it is impossible to produce tabes artificially in animals, it is certain that when bread contaminated with ergot of rye is habitually taken as food, posterior sclerosis becomes developed. Until lately the description of the symptoms which occurred in such cases has been somewhat obscure, and the results of inspection not sufficiently clear; but recently Tuczek has given a lucid description of an epidemic of this kind, which occurred in the district of Frankenberg (Hesse) subsequently to the bad harvest of 1879, when as much as ten per cent. of ergot of rye was found to be mixed with the flour of which the bread supplied to the inhabitants was made. The district mentioned contains twelve villages, with a population of 2,500; five hundred of the latter fell ill, many of whom were children. The common people there are miserably poor; the dwellings and the food are generally abominable; and, in addition to this, the people have an extraordinary proneness to dram-drinking, for on the average three quarts of raw potato-spirit are consumed daily by each person, and even children are found addicted to this pernicious habit. The harvest of 1880 contained much less ergot of rye than the one of 1879, and does not appear to have led to any fresh outbreaks of the malady; yet it gave rise to many relapses; and although the harvest of '81 was good, and the meal pure, cases nevertheless occasionally presented themselves even then for admission into the hospital, owing to relapses.

In some few instances the action of the poison appeared to be rapid, since severe cerebro-spinal symptoms were observed soon after the ingestion of the poisonous bread. In by far the largest majority of cases, however, the mental affection, as well as the epilepsy and tabes, broke out some months afterwards, and in some instances ergotism set in a considerable time after the consumption of the bread had been discontinued.

This seems to show that there is, apart from the primary affection, a kind of secondary or late intoxication—resembling somewhat the progress of syphilis—and leading eventually to severe disease of the centres of the nervous system. Seventeen patients suffering from ergotinic tabes were received into the University Hospital of Marburg, eleven being males and six females, including six children under fifteen years of age. There were other cases in which attacks of mania, with epileptiform seizures, formed the principal symptoms; yet in all cases without exception the knee-jerk was absent, and eventually returned only in a single instance, although a good many cases recovered. Even in children, in whom, as a general rule, the patellar reflex is so well marked, it could not be elicited after they had shown symptoms of ergotism.

In the cases of tabes, numbness, pins and needles, lightning-pains, a feeling of constriction round the waist, analgesia, staggering on closing the eyes, and ataxy of gait were present. The faradic sensibility was much diminished; and the faradic wire brush applied with a powerful current caused hardly any reddening of the skin. The pupils were large, as is often seen in the commencement of tabes. In four fatal cases Burdach's columns were found sclerosed, while Goll's columns appeared to be either normal or only slightly affected. The antero-lateral columns and the grey matter were healthy. In Burdach's columns the degeneration could be traced throughout the entire extent of these tracts, from the lumbar portion up to the medulla oblongata. The pia spinalis was found to be healthy. The changes in the cord consisted essentially of overgrowth of the neuroglia and atrophy of nerve-fibres. There were no traces of acute myelitis anywhere. Corpora amylacea were present.

The absence of the knee-jerk was of the greatest diagnostic value in all these cases. In some, where at first only epileptic seizures or mental symptoms were present, this symptom justified the conclusion that there was even then a definite anatomical alteration in the posterior columns of the cord. In one of these patients absence of the knee-jerk was the only sign of tabes, and yet the inspection showed that even at such an early stage there was decided evidence of degenerative atrophy in the cord. The blood-vessels and the pia mater were healthy, and so were the posterior roots and spinal ganglia ; but in the lumbar and lower dorsal cord the whole transverse section of Burdach's column was found to be diseased; further upwards only the middle portion of this system appeared to have suffered, while in the cervical cord only a small tract contiguous to Goll's column was found in a state of sclerosis.

A counterpart to ergotism is seen in the causation of spastic spinal paralysis, with presumably sclerosis of the pyramidal strands of the cord, by eating bread mixed with the meal of the grain of *Lathyrus Cicera.* This bread is consumed by the poorest classes in Upper and Central India, especially near Allahabad and in Upper Scinde, in Algeria, in some parts of France, and in the south of Italy, where the people call it "mochi." Hippocrates and Galen speak of *crurum impotentia* in those who fed on ervum, which is analogous to *Lathyrus*. Targioni-Tozzetti observed an epidemic of this disease, which he called "epidemia di storpio" (cripple-plague), in the last century. In 1873, Professor Cantani, of Naples, proposed to call it Lathyrism, in order to point out its analogy with ergotism. It was, however, Brunelli,[1] of Rome, who recognised that the peculiar affection caused by eating *Lathyrus* shows the symptoms of spastic spinal paralysis. He happened to see, in October, 1880, five cases of this kind, and found that all the patients had come from the commune of Alatri, near Rome; they were small farmers, three of whom belonged to the same family; and they had for several months been obliged to subsist on bread consisting of equal parts of rye and *Lathyrus*. Proceeding to Alatri, Brunelli found six more persons similarly affected. Most of them were young men under thirty-five years of age. The aged and the children who partook of the same bread did not appear to suffer. In that part of the country the disease had not been known before 1875, when a large quantity of *Lathyrus* grain had been imported and sold at a very low price.

The first symptom of illness consisted of debility and tremor in the legs; the patients had the appearance of being drunk, more especially after meals taken when the *Lathyrus* bread had been the only food. If the use of the bread was then discontinued, the people got quite well again; but in those whom poverty obliged to go on with it for any length of time the disease made rapid progress, and after two or three months presented the characteristic aspect of spastic paralysis. There was great stiffness in the legs, and impaired power of walking; the steps were short, and the feet dragged on the ground; the legs were approached to one another by rigidity of the adductors of the thighs; the toes were contracted in flexion, and the heels lifted from above the ground by contraction of the gastrocnemii. Those most affected seemed when sitting riveted to the chair, and could only get up after repeated efforts. Crutches and ordinary walking-sticks were of little use to them; but they got on better with a long sort of an alpenstock, of which they grasped the upper end with both hands. Muscular nutrition was not impaired; there was no affection of sensibility or of the special senses, the sphincters, or the brain. The tendon reflexes were exaggerated, more particularly as regards the rapidity with which the leg was jerked forward on percussing the patellar tendon, and less so with regard to the extent of the movement produced.

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[1] "Transactions of the International Medical Congress," London, 1881. Vol. ii., p. 45.

It has been noticed in Italy that pigs, after feeding on the fresh plant of *Lathyrus Cicera* begin presently to drag their hind legs. The dried grain of the same plant, when taken by pigs, dogs, and rabbits, causes similar effects. Rabbits seem to die soon after they have been fed on it, while hogs resist the action of the poison much longer. Lathyrism is by some authors believed to be identical with beri-beri, but recent researches seem to make it probable that the paraplegia of beri-beri is not owing to spinal disease, but to acute multiple neuritis.

Mr. Barron, of Liverpool, has given me an interesting account of an epizootic in horses which lately occurred in that city, owing to their having been fed on Indian mutters, which are supposed to be the seeds of *Lathyrus sativus*. A team-owner began feeding his horses, which were seventy-eight in number, and amongst which there were four ponies, on mutters in the beginning of October last. They remained apparently well, although the men complained of slowness of the lateral movements of the horses in stables and difficulty in backing. To this, however, no attention was paid until the end of March last, when the weather was cold and damp, with easterly winds. At this time a horse was seized with laryngeal spasm in the street, and fell dead from asphyxia. Nine others have since died, with the same symptoms; and in all thirty-three have been affected,—no ponies, and only one mare. In one horse tracheotomy was performed for the laryngeal spasm. Mr. Barron has as yet had but one post-mortem examination, in which he was unable to get the medulla oblongata, but found complete atrophy of the left recurrent laryngeal nerve and the muscles supplied by it; no apparent wasting of other muscles; but this remained doubtful, as microscopic examination of the right recurrent nerve and the muscles animated by it showed a commencement of atrophy. He also found wasting of the motor cells of the anterior cornua of the cord, which was most marked in the left side, with sclerosis of the corresponding crossed pyramidal strand.

It would be interesting to ascertain in future observations of this kind whether it is chiefly or exclusively the abductors of the vocal cords which are affected, and whether the adductors remain healthy or are contracted. Laryngeal "crises" with fatal results have occurred in patients affected with tabes, but their occurrence in spastic spinal paralysis is problematical. An examination of the patellar tendon reflexes in the suffering horses would show whether the disease from which they suffered partook more of lateral or of posterior sclerosis; but the wasting of the ganglionic cells of the anterior cornua of the cord, which Mr. Barron discovered, goes some way to show that it may have been that form of spinal disease which is known as amyotrophic lateral sclerosis.

I now proceed to consider the influence of another poison which is far more subtle in its nature, more widely diffused over the earth, and more destructive of human life and happiness in its effects than any other, viz., the poison of syphilis. *Syphilis*, which begins as a blood-disease, and, if unchecked in its course, ends as a flesh-and-blood disease, has long been known to play an important part in the production of certain diseases of the brain, such as tumour, arterial thrombosis, and meningo-encephalitis. It is, however, only quite recently that evidence has been brought forward from different quarters showing that it lies at the root of most cases of tabes which come under our observation in practice; and I have no doubt that it will presently be found to have likewise a direct or indirect influence in the causation of other forms of primary sclerosis of the cord. For practical medicine this is one of the most important facts which have been elicited by recent researches, as it seems to open up to us a new prospect of our being able by energetic and long-continued treatment of the venereal distemper to prevent the outbreak of tabes in many instances, and to cure it, if the patient comes under care in the initial stage of the malady.

When it once becomes known throughout the profession that insufficient treatment of the earliest symptoms of venereal disease opens the door to the invasion of such an awful disease as tabes, which causes the most severe sufferings that men are called upon to endure, and which when fully established baffles all our therapeutical resources, it may surely be hoped that the treatment of primary syphilis, and of the earlier stages of secondary syphilis, will be carried out in a more systematic manner than is now frequently the case, and that every trouble will be taken to destroy that baneful and insidious poison of syphilis before it has taken a thorough hold of the system. Instead of telling the patient, as is now often done, that his primary affection is "nothing," and "will soon be well," his attention should be drawn to the necessity of a long and persevering treatment, if he values his health and happiness; and he should be plainly advised that it is very often in cases which have a mild beginning that years afterwards the most terrible consequences are apt to supervene. We fortunately possess specific antidotes to the syphilitic poison in mercury and iodide of potassium; but these can only exert their full effects if perseveringly used for many months from the very beginning of the malady. Thus only can we eventually succeed in preventing the evolution of some of the direst diseases to which the human species is liable.

The earlier observers of tabes did not recognise the connexion between that malady and the venereal poison. Thus we find that Romberg,[1] that sagacious physician, to whose clinical acumen we owe the first and truly masterly description of the more prominent symptoms of the disease, when discussing the causes of tabes, does not say a single about syphilis. Duchenne,[2] many years afterwards, noticed that several of his patients had had syphilis, and that this appeared to be the only rational or obvious cause of the ataxy; but he added that such a conclusion was hazardous, inasmuch as there was no novel or special symptom in those cases, apart from certain well-known signs of the venereal distemper. It is true that the pain complained of appeared occasionally to be more severe or was only felt at night, but this also happened when there was no syphilis; while with regard to treatment, anti-syphilitic remedies had no beneficial influence on the disease. Fournier was the first who, in 1876, expressed himself decidedly for the syphilitic origin of tabes, and has more recently developed his ideas fully in a work[3] which, although written in the spirit of a special pleader, contains, nevertheless, a large amount of irrefutable evidence establishing his thesis. He was followed in France by Vulpian,[4] who placed syphilis in the first rank of those influences which determine the outbreak of the malady, and asserted that there are few patients suffering from tabes who have not, a few years before the appearance of the first symptoms, had an infecting sore and secondary syphilitic manifestations; that a few of these have been properly treated, but most of them very insufficiently so; and that amongst twenty patients suffering from tabes there were at least fifteen who had previously been syphilitic. Caizergues,[1] who has reported a hundred cases of syphilitic affections of the cord observed by various authors and himself, has from his own experience recorded three cases of undoubted syphilitic tabes. Grasset[2] has recently made the inspection of one of these latter cases, and found in the brain diffuse lesions of meningo-encephalitis, and in the cord systematic sclerosis, which, below the cervical enlargement, affected the whole of the posterior columns, and, above that point, only Goll's columns. In that case the ataxy had been confined to the lower extremities, but the patient had also suffered from general paralysis (megalomania). According to him, the circumstance that syphilis habitually produces diffuse lesions in the brain does by no means show that it could not produce systematic lesions in the cord; and he is of opinion that clinical evidence should, as far as this point is concerned, be held of greater weight than pathological anatomy.

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[1] "Lehrbuch der Nervenkrankheiten," Berlin, 1840.

[2] "De l'Electrisation Localisée, etc.," p. 655. 3rd Edition. Paris, 1872.

[3] "De l'Ataxie Locomotrice d'origine syphilitique." Paris, 1882.

[4] "Maladies du Système Nerveux," p. 245. Paris, 1879.

In this country, Moore, Dreschfeld, Drysdale, Gowers, Hutchinson, and others have expressed themselves in favour of the syphilitic origin of the disease in the majority of cases.

A few years ago it occurred to me[3] that it would be worth while to ascertain the influence of syphilis in the production of other diseases of the nervous system, and to compare the numbers thus obtained with those which had already been recorded for tabes. In this way a kind of cross-experiment would be made whereby I thought that a good deal of light might be thrown upon this question. I therefore analysed a thousand consecutive cases of nervous affections recorded in my case-books with regard to this point, without making the least attempt at selection.

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[1] "Des Myélites Syphilitiques," p. 72. Montpellier, 1878.

[2] "Traité Pratique des Maladies du Système Nerveux," p. 1017. 2nd Edition. Montpellier, 1881.

[3] "The Lancet," 1881, vol. ii., p. 496; and "Transactions of the International Medical Congress," 1881, vol. ii., p. 38.

Amongst these there were 206 of epilepsy; 101 of neurasthenia, without evidence of substantial lesions of the nervous system; 77 of hemiplegia owing to cerebral hæmorrhage or softening; 51 of neuralgia; and 32 cases of tabes with fully developed symptoms; the remainder were cases of hysteria, infantile paralysis, local paralysis, muscular atrophy, anæsthesia, chorea, tumour of the brain, impotency, paralysis agitans, torticollis, etc. It then appeared that in 29 out of the 32 cases of ataxy there was a syphilitic history; and in these 29, secondary symptoms had occurred in 28, while in one of them there had been a soft chancre and a bubo, but no secondaries.

This shows a percentage of 90·6 in favour of the syphilitic origin of tabes, which appears exceedingly high when compared with the percentages found for other nervous affections, inasmuch as of 206 cases of epilepsy only 10, of 101 cases of neurasthenia only 12, of 77 cases of hemiplegia 5, and of 51 cases of neuralgia only 2, had been preceded by syphilis. The percentages are, therefore, as follows:—

Tabes	was preceded by	syphilis in	90·6	per cent.
Neurasthenia	,,	,,	11·8	,,
Hemiplegia	,,	,,	6·2	,,
Epilepsy	,,	,,	4·8	,,
Neuralgia	,,	,,	3·9	,,

There were six additional cases in which paralysis of the ocular muscles, shooting pains, and sexual debility rendered it probable that sclerosis was developing in the posterior columns of the cord, and in four of these there were syphilitic antecedents. These cases, however, were lost sight of before the symptoms had become unequivocal; and, as they all occurred some time before the loss of the patellar tendon-reflex was utilised for the diagnosis of this disease, I think it better to exclude them. Yet it is a significant fact that in four out of these six doubtful cases syphilis should have previously occurred.

With regard to the interval which had elapsed between the first symptoms of syphilis and of tabes, it was found that the former had preceded the latter upwards of twenty years in two cases, between ten and twenty years in seven cases, between two and ten years in nineteen cases, and eighteen months in one case. Amongst the three cases in which there was no history of syphilis, the affection was attributed in one to an operation for piles, in another to an accident in a tramcar, and in a third to exposure to wet and cold. The age at which tabes became developed was from twenty-one to forty-five, and all the patients were males. In all cases, some other cause, such as accidents, over-exertion, the influence of wet and cold, and sexual or alcoholic excesses, were mentioned as having led to the outbreak of the complaint.

Since taking these percentages I have had thirty-four new cases of fully developed tabes under my care in private practice.[1] Amongst these patients twenty-eight acknowledged having had syphilis, while six denied any venereal infection. The percentage of syphilitic tabes in this set of cases would be therefore 82·4, and of the non-syphilitic 17·6. I think it right to add that amongst the six patients who denied syphilis, there were two who had, in consequence of the peculiar circumstances in which they were placed, the strongest possible inducement to refer the disease to accidents, and to deny any venereal infection. One of them had a suspicious papular eruption on the chest.

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[1] I have purposely abstained from utilising my hospital experience of tabes for the elucidation of this subject, as I have found the statements of hospital patients with regard to previous infection in general to be unreliable.

The entire percentage in the two sets of cases would therefore amount to 86·5 for syphilitic and 13·5 for non-syphilitic tabes.

In Germany it was chiefly Erb[1] who made himself the champion of the syphilitic theory of tabes. He found that, out of 122 cases which had occurred in his practice, syphilis had preceded tabes in 89 per cent. To these he added more recently another set of cases, bringing the whole up to 200. In the latter cases the percentage amounted to 91. In twelve hundred cases of other nervous affections the percentage for antecedent syphilis was only 22·75, while 77·25 had presented no syphilitic manifestations. Erb was therefore led to the conclusion that few people who have not had syphilis are likely to get tabes. Leyden,[2] on the contrary, states in a somewhat peremptory manner that the theory is wrong, because it is based on statistics only, and that statistics may prove anything. He considers that the anatomical changes found in tabes have not the slightest resemblance to other notorious lesions of syphilis, and that specific treatment does no good in tabes. For these reasons he rejects the specific theory of the disease; yet in the same paper he states that iodide of potassium must, after all, be looked upon as the most useful remedy for it! Westphal,[3] whose opinion is entitled to the highest respect, has likewise been, from the beginning, a consistent opponent of this theory. He has no faith in statistics, as he finds it impossible to rely on the statements of patients. Some of these latter consider any sore on the sexual organs to be venereal, and even surgeons are in the habit of treating any abrasion on those parts as syphilitic in order to be on the safe side.

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[1] "Centralblatt, etc.," 1881, p. 195; and "Transactions of the International Medical Congress," 1881, vol. ii., p. 32; and "Berliner klinische Wochenschrift," No. 32, 1883.

[2] "Tabes Dorsalis," in Eulenburg's "Encyclopädie, etc." Wien und Leipzig, 1883.

[3] "Archiv für Psychiatrie." Berlin, 1879-83.

Where opinions amongst those best qualified to judge differ so widely, a full discussion of all the different points in dispute appears to me the only way to arrive at the facts of the matter.

1. With regard to statistics, it seems hardly fair to exclude them altogether, as they are constantly utilised for determining various points in the pathology of other diseases, such as fevers, consumption, epilepsy, etc. To my mind the adversaries of the syphilitic theory of tabes would combat this view more effectually if they would furnish us with statistical data from their own practice, showing that syphilis has been absent in a large proportion of cases. All the percentages which have been published are in favour of syphilis being of great influence in leading to the appearance of the disease. They are as follows :—

Althaus ...	86·5	Gowers ...	70
Bernhardt	58·8	Pusinelli ...	52
Buzzard ...	45	Quinquano	100
Erb ...	90	Ross ...	95
Fournier ...	91·45	Vulpian ...	75

This surely cannot be a simple coincidence ; moreover, if all the facts were known, it seems probable that these percentages would appear even higher than they actually are. It is well known that syphilis is often overlooked by careless persons, where its manifestations, whether primary or secondary, are mild. A small primary sore, which heals readily in a short time, may be followed by a few spots of roseola and a slight soreness in the throat and tongue. This disappears with or without any treatment, and the patient thinks that he never had syphilis ; yet four, five or ten years afterwards double vision and a severe attack of lightning pains may herald the invasion of tabes, which is as plainly owing to the venereal disease as gummata or sarcocele.

2. It is frequently noticed in practice that patients deny having ever exposed themselves to infection, at a time when they show the most evident signs of it. More especially in hospital we often see patients who are covered all over with a syphilitic rash, and have ulceration in the throat and palate, and yet state plainly that they never had any "venereal." Even private patients, who ought to know better, occasionally suppress the information which is so important for us, from a sense of false shame. Some no doubt forget it in the course of time ; others overlook it ; and where there has been a urethral chancre or inherited syphilis, patients may be well excused for not knowing that they ever had such a complaint.

Case 1.—In August, 1879, Dr. Wright, of Derby, asked me to see a commercial traveller, aged fifty-three, who had been twice married, and acknowledged having exceeded a good deal in his sexual relations. His habits had been irregular. Three years ago his present illness commenced with a feeling of numbness in both hands. When he put his hands into his pockets to feel for anything, he had not the least notion of what there was there. I made him close his eyes, and put coins and other familiar objects into his hands, but found him totally unable to determine their nature. He could, however, still feel sharp pricking and pinching. He had optic atrophy in both eyes to such an extent as to be unable to read No. 19 of Jaeger's text-types. Argyll-Robertson's symptom was present. The hearing on the left ear was defective. He complained of pain in his legs, which was sometimes acute and darting, at other times dull, and was frequently confined to a small area in the leg not larger than a crown-piece. The patellar reflex was absent in both sides. He was very unsteady on his legs ; could not stand on one, without being supported and with his eyes open ; nor could he balance himself on both legs with his eyes closed. The gait was ataxic in the highest degree ; he had the greatest difficulty in going downstairs, and in the dark he could not walk at all. The spine appeared to be tender on pressure. The bladder was sluggish ; the sexual power and desire very much diminished ; on attempting copulation, erection was imperfect, and ejaculation premature. He had lately been often subject to small abscesses on the second finger of the left hand, which made the whole arm so tender that he was unable to use it. On being asked whether he had had syphilis at one time or another, he answered, "that he thought he had had *something*, but did not remember what it was ; yet he believed that a doctor had burned him."

In the following case, antecedent syphilis was at first denied, and subsequently acknowledged :—

Case 2.—In October, 1879, Mr. Bader asked me to see a merchant, aged forty-four, who had for three years past suffered from paralysis of the left rectus externus muscle and gradually advancing atrophy of both optic nerves. He could only just read No. 6 Jaeger ; had achromatopsia and bilateral myosis. He denied having ever had any syphilis, and attributed his illness to excessively hard work and annoyance in business. He also suffered from chorea, which had commenced during childhood and never left him ; and he had been impotent for five years. The patellar reflex was absent, and there was some ataxy in standing, but not in walking. After the patient had been under my care for some time, he one day told me that he had really had syphilis about ten years ago, and suffered for twelve months from various manifestations of the distemper, chiefly in the mouth and throat. He gave as his reason for denying this, in the first instance, that "the very thought of his foolish behaviour at that time annoyed him so much that he preferred not to talk about it."

Case 3.—In April, 1879, I was consulted by a merchant, aged thirty-five, married, and father of four children, who attributed the symptoms of tabes from which he was then suffering to an accident which he had had in a tram-car, Ten months ago he noticed that he suddenly saw everything double ; and the right eye was turned inwards, so that he evidently had had paralysis of the right rectus externus. He recovered from this in about three months. When I first saw him there was a degree of ptosis in the right eyelid, making the right eye look considerably smaller than the left. The knee-jerk was absent in both sides. Walking was troublesome, the right leg being worse than the left. He could stand pretty well on the left leg, but could not balance himself when standing on the right. There was a tight feeling across the chest and throat, and great numbness from the waist downwards. I asked him whether he had had syphilis, and he denied it indignantly. On examining the penis, however, I discovered an enormous scar on the gland. To my question as to the origin of this scar, the patient replied that it was owing to the resistance he had experienced on consummating marriage ! It had, however, all the appearance of a syphilitic cicatrix ; and the patient eventually recovered completely under specific treatment.

In some cases the initial symptoms of syphilis are so very slight that a lay patient might have some difficulty in believing that so little could have led to so much ; while the medical intellect is better able to grasp the full bearings of this influence.

Case 4.—In September, 1882, I was consulted by an Irish practitioner, aged forty, single, who told me at the beginning of the interview that twenty years ago he had had a very mild form of syphilis, namely, a small chancre which healed rapidly, and six weeks afterwards a slight ulceration in the tongue. He had never had any rash on the skin, or ulceration in the throat, or any further manifestation of the specific diathesis ; and he had remained apparently in very good health *for fifteen years*. Five years ago, however, the first symptoms of his present illness appeared, and affected more particularly the right side of the body. Dimness in the vision of the right eye came first, and optic atrophy is now well established. Lightning pains in the right knee then supervened, and are at present apt to come on in almost any part of the body. They last a few hours, and then vanish, generally under the influence of chloral. Eight or ten days afterwards there is another bout of pain. There is a slight degree of ptosis of the right eyelid, but no palsy in the muscles which move the eye itself. Great numbness is felt in the sphere of the right ulnar nerve. The sexual power and desire are gone. Twelve months ago he had the last imperfect connection, but he still has occasionally seminal emissions during sleep. The bladder is sluggish ; incontinence is not unfrequent ; the bowels are constipated. The knee-jerk is absent in both sides, and the muscles of the lower extremities are much wasted. The patient staggers when standing with his eyes closed and in the dark ; and the gait shows well-marked ataxy. He had never been able to take iodide of potassium ; and had just returned from Aix la Chapelle, where he had had forty mercurial inunctions without the least result.

It may be safely assumed that, if this patient had not been a medical man, he might have forgotten all about the very slight specific affection which had preceded the outbreak of tabes by fifteen years. As it was, however, he was quite convinced of the syphilitic nature of his affection, as he could not think of any other cause which might have led to his present illness.

Case 5.—In August, 1881, Dr. Meurer, of Wiesbaden, sent a gentleman to me who was forty-two years old, married, and a great sportsman. Some years ago he noticed that he saw everything double, and had, therefore, great difficulty in shooting. This arose apparently from paresis of the right rectus internus. When I saw him, that muscle was quite paralysed, and there was also ptosis and paralysis of the rectus superior of the right eye. The tendon reflexes in the knee were absent, and the patient experienced the characteristic difficulties in walking and standing. On my asking him whether he had had syphilis, he said that twenty-five years ago he had had a " soft sore which lasted only two days " ; that he had swallowed large doses of iodide of potassium, and had undergone a long treatment at Aix-la-Chapelle without the slightest benefit ; and that he did not consider that he really ever had had syphilis.

Fournier's experience with regard to this particular is that tabes generally becomes developed where the early symptoms of syphilis have been unusually mild. Amongst eighty-four cases in which this point was particularly inquired into, there was not one of severe syphilis ; ten were of medium intensity, twenty-one mild, forty very mild; in ten there had been a chancre without subsequent secondary symptoms ; and in three the patients were unaware that they had had syphilis. He says that most of his tabid patients have only had a slight rash on the skin, insignificant erosions of mucous membranes, alopecia, and swollen lymphatic glands ; and that all these symptoms disappeared rapidly with or without treatment. A natural consequence of this is that such cases are insufficiently treated. Of seventy-nine such patients, two had undergone no treatment at all, forty-six had had treatment extending from a fortnight to four months, while thirty-one had had a longer treatment. It is, however, well known that syphilis cannot be cured by a short treatment, which latter does little but suppress its manifestations for the time being.

In other cases, however, the syphilitic symptoms are prolonged and severe, and become so intertwined with those of tabes, that no one would be able by any amount of sophistry to explain away the connection between the two diseases.

Case 6.—In January, 1883, I saw a patient in consultation with Dr. Mullar, of Kilburn, who was aged thirty-one, a baker by trade, married and childless, and who had had a hard chancre and a bubo, quickly followed by secondaries, six years ago. Ever since that time he had suffered from some constitutional manifestations of the syphilitic dyscrasia, being never thoroughly well. Six months ago, sarcocele became developed in the left testicle. Ten days ago he had a severe epileptic fit, which lasted five minutes; and the day before I saw him he had had another, in which he passed the excretions under him. His memory had of late become much impaired; he was unable to attend to his business, in consequence of feeling always muddled and confused. The knee-jerk was absent on both sides, and the patient staggered when standing with his eyes closed. I saw him again in the following October, when some additional symptoms of tabes, viz., paralysis of the left rectus externus and lightning pains, had made their appearance. The mental hebetude was more marked, and alternated with violent outbreaks of temper. On one occasion he made an indecent proposal to his nurse, in the presence of several other people; then threw himself on the floor, where he rolled about on all fours, and swore in such a frightful way that he terrified all those present. The symptoms of tabes were in this case evidently being developed together with those of general paralysis of the insane; and meningo-encephalitis, combined with sclerosis of the posterior columns, would most probably be found, were it to come to an inspection.

Case 7.—About the same time I saw, in consultation with Dr. Grasemann, a patient who was an auctioneer, aged forty-four, married, but childless, and who had had a somewhat severe form of syphilis ten years ago. There had been an indurated chancre and a bubo, followed by bad and prolonged secondary symptoms. He had altogether lived a wild life, masturbated as a boy, committed venereal excesses later on, and had drunk and smoked heavily. He had suffered from diplopia some time ago; and five years ago had an attack of aphasia and right hemiplegia, from which he recovered in a few days. Several such attacks had taken place since then at intervals, leaving no traces after a short time. The last hemiplegic attack occurred only five days ago, and then affected the *left* side of the body. The memory was much impaired, and the patient felt occasionally so confused that he was unable to attend to his business. When I saw him, the hemiplegia of the left side had already disappeared. There was, however, total loss of knee-jerk, Romberg's symptom, constipation of the bowels, sexual excitement and debility, and occasional attacks of spasmodic incontinence of urine, thus rendering the diagnosis of tabes certain.

Occasionally tabes results from direct inoculation of the syphilitic virus.

Case 8.—A well-known and able surgeon consulted me in May, 1871, and informed me that, while examining a woman in a workhouse infirmary in 1866, he inoculated the index of the right hand with syphilis. For two years afterwards he was, to use his own words, never free from that wretched disease in one form or another. He then became apparently quite well, and continued so until Christmas, 1870, when he was rather suddenly seized with tingling in the legs and numbness in the left arm and side of the body; a day or two afterwards double vision supervened, and there was a feeling of constriction round the waist and inability to walk. Some of the medical friends whom he consulted thought the symptoms owing to the extreme cold which prevailed at that time, while others believed them due to syphilis; and there was, therefore, some hesitation how to treat it. However, he slowly but steadily improved, and was able during the last six months to resume his practice. He now complained chiefly of more or less constant constriction round the waist, which was at times almost unbearable. His left arm and both legs were numb and stiff. He could not balance himself with his eyes shut, but would fall backwards unless supported. He also had sudden shocks of stabbing pain in the lower extremities, and on awaking in the morning felt a curious wave of sensation flowing down the left arm and up the legs towards the perinæum.

3. Several observers have expressed the opinion that the morbid anatomy of tabes speaks against the syphilitic origin of the disease. Thus Chauvet[1] says "that syphilis can never cause a primary sclerosis of the posterior root-zones." Lancereaux[2] considers that the lesions of tertiary syphilis never invade an organ through its whole extent; they are, according to him, circumscribed, forming single or multiple nodes grouped in one or several spots, which undergo a granular, fatty, or hyaline degeneration, and terminate in loss of substance or a cicatrix. The lesions of tabes, on the other hand, are diffuse, extended, systematic, and although slowly developed, like those of syphilis, do not produce loss of substance, nor do they leave a scar. Westphal expresses a similar opinion; yet I do not hesitate to say that no one, however experienced, is able by inspection alone, even if aided by the microscope, finally to decide what is syphilitic or not. Moreover, we are familiar with diffuse changes occurring not only in the blood-vessels, but also in the liver, kidneys, and other organs, and which are generally acknowledged to be owing to syphilis. Diffuse acute hepatitis and acute glossitis may be of a specific nature, and affect the whole, or almost the whole, of the liver or tongue. Lancereaux's statement, that tabes does

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[1] "Thèse de Paris," No. 55. Paris, 1880.

[2] "Transactions of the International Congress, London, 1881." vol. ii., p. 40.

not produce any loss of substance, seems to be somewhat of a solecism ; for what is wasting but loss of substance ?

On looking through pathological literature we find all and every kind of lesion in the spinal cord described by reliable observers as arising from syphilis. Cases are on record of acute myelitis, either diffuse or limited to certain portions of the cord, such as the lumbar enlargement, the lumbo-dorsal, dorsal, and cervical portion ; this again may be transverse, superficial, or central. Then we find cases of acute ascending spinal paralysis without any appreciable anatomical lesion, and of all the different forms of chronic myelitis ; while all the different systems of the cord, including the anterior horns, have been seen to be separately or collectively affected. If this be true—and any one may convince himself that it is by searching the vast storehouses of the records of morbid anatomy—then we can certainly not accept the statement *ex cathedrâ,* that syphilis *cannot* produce sclerosis of the posterior columns. The principal lesions of tertiary syphilis are gummata and sclerosis. The hard chancre, to begin with, is a form of sclerosis ; and the "cirrhosis" which is found in the tertiary period in the rectum, the liver, lungs, and kidneys is nothing but sclerosis.

The objection that syphilis never produces any system-disease elsewhere is surely untenable. We know that the distemper often attacks exclusively such systems as the skin and hair, the lymphatic glands, and the periosteum. As the syphilitic virus is one of a highly specialised character, it seems only rational to expect that it should affect certain systems in preference to others. Nobody doubts that the influence of cold may produce tabes ; but it is certainly more difficult to understand how a general cause, like cold, should set up morbid changes in special systems than that a special poison like syphilis should do so.

Quite apart from this, however, we have seen (p. 48) that tabes is not absolutely a system-disease confined to the posterior columns of the cord, but that almost invariably, besides sclerosis of those parts, posterior spinal meningitis, wasting of the posterior roots and the posterior cornua of the grey matter, and of Clarke's vesicular columns, is found, while the ganglionic cells of the anterior grey matter are not unfrequently seen to be wasted, and the cranial nerves sclerosed. We are therefore justified in stating that tabes is a much more general disease than is expressed by the term "sclerosis of the posterior columns."

The idea that something absolutely special ought to be discovered, showing a definite syphilitic origin, appears unreasonable, more especially if we consider that what is called the "classical type" of tabes is probably only rarely idiopathic, and much more frequently specific.

Finally, it will surely be acknowledged that where morbid anatomy and clinical medicine appear to clash concerning the nature of a disease, the latter science should carry the day. I may here fittingly refer to an interesting discussion which took place at a recent meeting of the Clinical Society of London[1] (October 26th, 1883), when I brought forward a case of apparently syphilitic tumours of the cerebral membranes, in a patient in whom there had been no history of congenital or acquired syphilis during life. The anatomical appearances in that case were as characteristic as possible for syphilis; yet there was an almost unanimous expression of opinion on that occasion that *anatomical structure without life-history is, in a general way, insufficient for a diagnosis*, and that the tumours which were described might have been owing to the tubercular or some other dyscrasia.

4. Another objection which is frequently made to the syphilitic theory of tabes is, that *we do not find any special symptoms in syphilitic tabes* which would distinguish it at a glance from ordinary tabes. I have already mentioned that it was this circumstance which deterred Duchenne from pronouncing

[1] "British Medical Journal," vol. ii., 1883, p. 874.

himself more strongly for the syphilitic origin of the disease. If we consider, however, that the description of the symptoms of tabes is in general taken from cases of syphilitic tabes, we can no longer be surprised at that circumstance. Moreover it is obvious that such an objection, if allowed, must also apply to other forms of tertiary disease, more especially to that which is apt to occur in the brain, and which is by general consent taken to be syphilitic. An attack of hemiplegia from disease of the cerebral blood-vessels may have, and often has, exactly the same symptoms as one arising from embolism or cerebral hæmorrhage, where there is not an atom of syphilis in the case. Some of the visceral complaints induced by syphilis do not produce symptoms which are very different from those owing to idiopathic disease of the same viscera. The symptoms of tabes should be looked upon as the response of the posterior columns of the cord to certain injurious influences; and this view explains why all symptoms which occur in non-syphilitic tabes may occur in the specific form of the disease, and *vice versâ*. The chief difference we find in the syphilitic form is, that there are not unfrequently other symptoms of venereal affection not referable to the cord.

5. This leads us to speak of another objection which has been made to the theory which we are now considering, viz., that in tabes we do not meet with any *other of the well-known symptoms of syphilis*. To this I would reply, that if such symptoms are properly looked for, they are frequently discovered; they were very striking in one of the cases which I have just related (No. 6), and to which I could add many others. Indeed, it is not at all uncommon to find primary or secondary cicatrices in the skin, loss of substance in the uvula, sarcocele, exostoses, and other notorious symptoms of syphilis in such patients; while evidence of brain-syphilis, more particularly of the congestive form of it, is likewise frequent. At the same time

no doubt cases occur where such symptoms are absent, more especially in the skin and mucous membranes ; but at this we cannot be surprised, if we consider that tabes is in general a tertiary manifestation of the syphilitic distemper. Similarly we find that in specific brain-disease hemiplegia, with late rigidity of the paralysed muscles, may be the only symptom ; while the clinical history of the case shows quite plainly that the nature of the affection is syphilitic. A very marked case of this sort, which occurred some time ago in my practice, is described in the " Transactions of the Clinical Society of London."[1] General paralysis of the insane frequently ends the career of the tabid as well as of some syphilitic patients.

6. A final objection which is frequently made to the syphilitic theory of tabes is, that the *malady is not cured by specific treatment.* A cure, however, of any affection which has already destroyed highly specialised structures is impossible under any circumstances. It is quite true that syphilitic patients daily recover their health under specific treatment ; yet no amount of mercury and iodide of potassium will restore a large hole that has been made in the palate, or the peculiar structure of a testicle which has undergone the process of sarcocele. Now it is well known that, even in the beginning, tabes is not a functional, but a structural disease (p. 42) ; and, in general, all we can expect therefore is to arrest the further progress of the complaint, and to cure or improve those symptoms which do not depend upon absolute destruction of nervous matter. Specific treatment is mostly useless when the second stage of the disease has been well established ; and likewise for the amblyopia and amaurosis of the first stage. *The more highly specialised the structures affected, the less likely is treatment to act beneficially ;* and this is no doubt the reason why optic atrophy constitutes such a hopeless subject for therapeutics.

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[1] Vol. xv., p. 203. London, 1882.

Syphilitic brain-disease is equally rebellious to specific treatment when it has once proceeded to destruction of specialised structures. No amount of iodide of potassium will ever cure softening of the motor zone of the brain, from thrombosis of the middle cerebral artery; yet the clearest possible connection may exist between the primary venereal disease and the subsequent affection of the cerebral blood-vessels.

Most cases of tabes which come under our care in practice have already lasted a number of years before they are recognised. Only too frequently lightning pains are put down to rheumatism or gout, and palsies of ocular muscles to exposure to wind. Yet many years ago Ricord said that the paralysis of an ocular muscle was the signature of syphilis on the eye of a patient! Fortunately we have now in the loss of the knee-jerk a diagnostic test of the utmost value for the recognition of the earliest beginnings of tabes, and one which will no doubt lead in course of time to an immense improvement in the therapeutics of this disease.

While, therefore, confirmed cases of syphilitic tabes, where the disease has to a great extent run its course, cannot be cured by specific treatment, the latter is very useful as long as an irritant morbid process is still actively going on in the cord. *The newer a symptom, the more likely it is to yield* to mercury or iodide of potassium; yet even old symptoms, if occurring in less highly specialised structures, are often greatly benefited. This is more particularly the case with the lightning pains, which may yield to the hydrargic perchloride after having resisted subcutaneous injections of morphia. The further progress of the disease may also be often arrested; and patients, who were fast going down hill, often regain at least some degree of health and comfort by a specific treatment. The patient whose case is referred to on p. 84 recovered completely. Rumpf, of Bonn, has cured a man who was in the second stage of tabes, by mercurial treatment continued for eight months consecutively. Hammond reports a similar case. Palsies of ocular muscles are now often in the very beginning treated energetically on a specific plan, with the result that the patients not only recover from such palsies, but never develop other symptoms which may have been on the point of breaking out. Even Leyden, who utterly scouts the idea of any connection between tabes and syphilis, is compelled to acknowledge that after all iodide of potassium is the best medicine for this disease ! (*loc. cit.*, p. 46).

The time during which the syphilitic germs breed in the system before they can produce tabes varies in different individuals. The most frequent period of its outbreak is between six and ten years after the primary infection. In exceptional cases it is much earlier, and I have now a patient under my care in the hospital, in whom it appeared a twelvemonth after a chancre :—

Case 9.—In April, 1883, Dr. Shepard, of the Euston-road, asked me to see an artist, aged forty, single, who had contracted syphilis from a model eight years ago. *Twelve months afterwards* undoubted symptoms of tabes appeared, as the patient found great difficulty in standing and walking, and showed the characteristic gait of ataxy. He likewise suffered from attacks of lightning pains, which were, however, not of a severe character, and appeared at long intervals. He was treated by iodide of potassium, which did no good at all ; after that he was subjected to inunction with mercurial ointment, and given blue pill, which did good for a time, but then seemed to lose its effect, as the disease gradually gained upon him. When I saw him, he was just able to walk a few steps on level ground with the aid of two sticks ; showed the classical gait of ataxy, and had lost his patellar reflexes. He could hardly stand with his feet wide apart and his eyes open, and would have gone down at once if the feet had been approached to one another and the eyes closed. Yet he had no difficulty in crossing one leg over another, and showed considerable muscular force in resisting attempted flexion of the knees. The brain and cranial nerves were healthy. He suffered from obstinate constipation, and the sexual power was completely lost. There was numbness in the soles of the feet, but in all other parts of the body sensibility was perfectly normal. As neither his eyes nor his hands suffered in any way, he was able to paint almost as well as before the beginning of his illness, the only thing which he could not do being to take a perspective of his pictures by walking backwards from his easel.

Case 10.—Another case in which the symptoms of tabes appeared early after syphilis was that of a merchant whom Mr. Bader sent to me in February, 1880. This patient was thirty-four years old, married, with three children, and had syphilis and gonorrhœa ten years ago. *Twelve months afterwards* lightning pains had appeared, which affected more or less all parts of the body. Eighteen months ago his sight began to fail : he has now Argyll-Robertson's symptom and amblyopia from atrophy of the optic nerve. The patellar reflex is absent. The legs feel as if they did not belong to him. The sexual power is diminished ; yet his wife is in the family way, and he had had connection a fortnight ago.

On the other hand, the patient may remain for many years in apparently good health after he has contracted syphilis ; so that the appearance of the first symptoms of tabes is believed to be unconnected with the specific taint, and put down to other causes.

Case 11.—Such was the case of a gentleman, aged forty-one, single, whom I saw at the request of Mr. Bader, in August, 1883. He had masturbated heavily at school, and contracted syphilis ten years ago. The symptoms of this illness were slight and of short duration, and the patient had been in thoroughly good health until about three years ago, when his sight commenced to become dim.

This was at first believed to be a local affection, and treated as such until Mr. Bader suspected it to be part and parcel of a general disease. I found, on examination, loss of knee-jerk, Romberg's symptom, constipation, difficulty of micturition, and a history of lightning pains, rendering the diagnosis of tabes certain.

In the following case, the interval between the occurrence of the primary sore and the appearance of the first symptoms of tabes was *seventeen years.*

Case 12.—In May, 1883, I was consulted by an army surgeon, aged forty-two, married, with three children, who had had syphilis badly *twenty years ago,* but had been apparently quite well afterwards. Three years ago lightning pains appeared in the legs, arms, and head ; and soon afterwards considerable difficulty and awkwardness in walking was experienced. At present he can walk for about fifteen minutes at a time, after which he is thoroughly done up. There is numbness in the legs and in the sphere of both ulnar nerves. Romberg's symptom is well marked. "Belt-sensation" is troublesome ; and the knee-jerk is absent in both legs, which are much emaciated. The patient has considerable difficulty with his bowels, but none with the bladder. The sexual power is diminished, yet there are not unfrequently nocturnal emissions of semen.

The length of time which intervenes between the outbreak of the two diseases does not speak against their standing in the relation of cause and effect. In other diseases many years are known to elapse before a cause produces an effect. Sir Benjamin Brodie met with an accident while riding on horseback in 1834, dislocating his right shoulder, and he eventually died of cancer which had become developed in the same joint in September, 1862, which gives an interval of twenty-eight years between the injury and the consecutive disease. It would not be difficult to multiply such examples. Moreover, no one thinks of denying that manifestly syphilitic symptoms may and do occur in the bones, testicles, and even in the skin, as long as twenty or thirty years after the primary affection.

Some of those observers who are fond of attempting to reconcile contradictory opinions have started the idea that syphilis acts *indirectly* in producing tabes, and is what is called a predisposing cause. It is said to lower the system, to deprive it of its power of resistance, and in this way to act like other debilitating influences, such as masturbation, sexual and other excesses, etc. This view, however, is hardly supported by what we see in practice. In many cases, indeed, so far from being debilitated by the germs which are breeding in them, the patients appear to be quite well and strong at the time of the outbreak of tabes, and this more especially where the primary and secondary symptoms have been slight.

Case 13.—A merchant, aged fifty-one, married and father of six children, consulted me in February, 1881. He had had syphilis twenty years ago, and his eldest child had been syphilitic. For many years past he had been perfectly well; but in March, 1880, he suddenly saw everything double. He consulted the late Mr. Critchett, under whose care the affection disappeared within a month. He had a second attack of the same thing in January, 1881, and was again subjected to treatment, but this time without any benefit. I examined him carefully for symptoms of tabes and syphilis, and could discover nothing wrong except complete loss of the knee-jerk in both sides and paralysis of the rectus externus muscle. His general health was excellent; yet there could be no doubt that he was in the first stage of tabes.

It is a curious circumstance, and one that is of some moment in the question with which we are now occupied, that both tabes and syphilis are much more frequent in males than in females. Fournier finds that amongst syphilitic patients there is one woman to nine men; while the proportion in tabes is about one woman to ten men. Where tabes occurs in women, it is generally in the lower orders of prostitutes, or in poor women who have to work hard in damp shops. Erb has seen thirteen tabid women, of whom four had apparently not had any syphilis. Three others denied having had it; but the first of these latter had had three miscarriages, had suffered from violent headaches, and four of her children had died young. The second of the three had had two miscarriages, and showed cicatrices resulting from extensive ulcerations of the skin. The husband of the third person was notoriously syphilitic. Four others had certainly had syphilis; in another this was highly probable; one more had had a chancre. This is rather a heavy indictment, and more particularly so when we remember how difficult it sometimes is to discover syphilis in a woman, and how easy, on the other hand, to overlook it.

While, therefore, an overwhelming amount of evidence speaks for a causal relation between syphilis and tabes, we must acknowledge, on the other hand, that cases are occasionally met with in practice where tabes and syphilis occur together, and where, under the influence of specific treatment, the syphilitic symptoms on the skin and the mucous membranes are improved or cured, while the symptoms of tabes become worse. These may afterwards improve under the influence of electricity and nitrate of silver. Such cases are certainly startling; at the same time they only prove what is already known, viz., that in general an anti-syphilitic treatment has only little influence on tabes when this has become firmly established, that is, after the second or ataxic stage has been fairly reached.

In many cases no other cause but syphilis can be found for the evolution of tabes; while in others a variety of injurious influences appear to act together with the syphilitic dyscrasia in leading to the outbreak of the complaint. The principal one of these latter is unquestionably the influence of *cold*. Most observers are agreed on this point, and only Fournier appears to attach little importance to it. The influence of cold in the syphilitic appears occasionally to act as the exciting cause, while in the non-syphilitic it is often mentioned as the only cause which can be assigned.

Case 14.—In May, 1874, I was consulted by a patient, aged fifty-nine, married, who had for a good many years been in business in Canada as a provision-curer. He had there to pass constantly from one extreme of temperature to another, as he had first to go into ice-houses and afterwards into the sun, when the thermometer in the shade was at 90°. He had, however, also had syphilis and gonorrhœa eighteen years ago, and had a stricture of the urethra, which gave him a great deal of trouble. Three or four years ago he suddenly lost his speech and became paralysed on the right side. He was unconscious for a fortnight, and then recovered. At present he has the true gait of ataxy, difficulty in standing, numbness up to the waist, complete loss of sexual power, but not of desire, and constipation of the bowels. He can generally pass his water in a small stream, but has occasionally attacks of retention, when the catheter has to be introduced. The symptoms of tabes had become developed within the last three years, and were by the patient and his friends attributed to the chills he was in the habit of receiving by going into ice-houses. Yet we cannot ignore that he had had syphilis, and the fact of his having had temporary aphasia and hemiplegia, in addition to his other troubles, speaks strongly for the specific character of all his nerve-symptoms.

Case 15.—Another case in which the influence of cold appeared to act as the exciting cause of tabes in a syphilitic subject was that of a merchant, aged thirty, single, who consulted me in January, 1873. He had led a very wild life, and had syphilis badly four years ago. He consulted, amongst others, Professor Lewin, of Berlin, and was treated by him with subcutaneous injections of perchloride of mercury for a month, after which all symptoms of syphilis left him. He spent the autumn of 1870 at Brighton, and was advised to take sea-baths very late in the season. After this he first began to feel shooting pains in the lower extremities, more particularly the right leg, and had difficulty in walking, with sluggishness of the bladder and constipation of the bowels. Romberg's symptom was present. He remained for some time under my care and I had the opportunity of satisfying myself that the syphilitic taint was by no means eradicated from his system, as he would occasionally for weeks together have specific eruptions on different parts of the body, such as ulceration of the tongue and of the penis.

A singular case in point is that of a Russian officer who acquired a chancre while serving with the army in the Caucasus, and treated the sore with the application of what he called "snow-poultices"! Soon afterwards he felt a shooting pain in the groin, scrotum, sacrum, and left leg, which induced him to apply to a surgeon. He now underwent treatment by inunction, which appeared to be successful for the time. Three years afterwards, however, he was obliged to stand in the water for a couple of hours, and then felt almost at once the same pain in the left leg and other parts which he had had on applying the snow. Shortly after this exposure had taken place, double vision, constriction of the chest, and other symptoms of tabes supervened.

The influence of exposure to cold is no doubt to a great extent instrumental in causing the numerous cases of tabes which occur after campaigns. When the Austrians had lost the battle of Sadowa, they had in their precipitate retreat to cross the river Elbe ; and it is related that amongst those who had to flee for their lives, and were thoroughly wetted on that occasion, an unusual number of cases of this malady occurred. Romberg mentions that the disease was rife during the great French wars in the commencement of this century. A large number of cases appear also to have occurred in the celebrated free-corps commanded by Major Von Lützow in the German war of liberation, in 1813, when that troop of irregular horse underwent the most extraordinary fatigues in incessantly harassing the outposts of the French armies. After the war in Hungary in 1849, and the French campaign in Mexico, similar results have been observed. In campaigns, however, the influence of cold is only one of the factors; syphilis, over-exertion by forced marches, and excesses of various kinds play, probably, quite as important a part in the production of the disease as cold.

In ordinary every-day life the influence of cold in producing tabes is observed chiefly in persons who sleep in newly built houses, or out of doors, or in damp bedrooms; who work in cold, draughty workshops or offices, or as agricultural labourers in the fields. Tabes seems occasionally to spring from frost-bite. Exposure to cold also has a most prejudicial influence after the disease has become developed. It often gives a fresh impetus to the morbid process, and renders the patients more useless and helpless than they were before.

Case 16.—This circumstance was well shown in the case of a merchant, aged forty, a widower, who consulted me in October, 1873. He had had gonorrhœa many times, and syphilis twenty years ago. He had infected his wife and only child. About twelve months ago he had a sudden attack of double vision, and began to suffer from spermatorrhœa and lightning pains through the legs. He consulted a quack, who told him that "his blood was too hot," and advised him to walk with his naked feet on the bare floor for half an hour every evening. After the patient had done this a few times, he rapidly lost the use of his legs, and was now totally unable to walk or to stand, but managed, with crutches, to walk a maximum of three hundred yards. He still could, when sitting on a chair, cross one leg over the other, and move the legs in bed quite easily in all directions. The bladder was extremely sluggish, the bowels confined, and the soles of the feet completely benumbed.

The next cause which is of influence, as exciting the development of tabes, is *over-exertion*. Experimental over-stimulation of the muscles in animals, by long-continued faradisation, has led to paralysis of their limbs, as seen by Vulpian, Brown-Séquard, and others; and it is easy to understand how over-exertion, either in walking or other ways, should exhaust the vital power of the spinal cord. This is more particularly the case with the syphilitic, while in otherwise healthy persons it is more rare; yet it was the only cause which could be discovered in the following case:—

Case 17.—A merchant, aged fifty, consulted me in November, 1871. He had for a number of years been engaged in mining operations in South America, which entailed a great deal of going down on ladders and crawling on niches in rocks, at a depth of upwards of a thousand feet. He first felt weakness in the legs seventeen years ago, after he had done "an unusually hard job" of this kind. He went on with his work, however, but gradually got worse, and about four years ago was recommended to take hot mineral baths in South America, after which the disease appeared to make more rapid progress. When I saw him, symptoms of tabes were marked in both upper and lower extremities, and there was also a considerable amount of wasting in the muscles of the hands. He had never had syphilis.

The following is another case in point:—

Case 18.—In June, 1874, I was consulted by a young gentleman, aged twenty, who was then reading for the bar, and was chicken-breasted and hunchbacked, but had been pretty well until the summer of 1873, when he went to the Vienna Exhibition, where he was walking and standing about all day long. After some time spent in this manner, he began to feel great numbness in the feet and legs, which gradually spread upwards to the waist. He was also very much troubled with a feeling of tightness round the chest. Symptoms of catarrh of the bladder, with great irritability of the viscus, then appeared, and the patient gradually got so weak on his legs that he had to give up walking altogether. At present he was totally unable to walk without assistance, yet had not the least difficulty in moving his legs in bed, and showed considerable muscular force in resisting attempted flexion of the knees. He had completely lost the power over the bladder, and was obliged to introduce the catheter sometimes every hour, more especially in the night, when he could never sleep properly on account of constant calls to pass his water. The urine was ammoniacal. and contained a large quantity of muco-pus. There were no symptoms of tabes above the waist. The patient denied having ever suffered from gonorrhœa or syphilis, or been much exposed to cold, and attributed his malady entirely to the over-exertion which has been mentioned.

Accidents appear occasionally to lead to tabes, without any syphilitic infection or any other cause having previously occurred. This question has recently been studied by Petit,[1] who has collected forty-seven cases, and concludes that injury to the spine, whether direct or indirect, by falls on the back, the seat, or the feet, may cause a concussion of the cord, and subsequently lesions, which may become the starting-point of a chronic myelitis, showing the symptoms of tabes. He thinks that more especially in persons predisposed to sclerosis in general, such as the gouty, the syphilitic, and the alcoholised, injuries at a distance from the spine may over-excite the cord, and lead to the development of tabes ; that they may aggravate the existing disease, or cause a relapse where it has been cured.

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[1] "Revue Mensuelle," Paris, March, 1879.

I am inclined to agree with Petit's views, from having had several cases under my care in which a severe accident was the only cause which could be ascertained.

Case 19.—In June, 1882, I had a patient, aged fifty, under my care at the hospital, who had been a digger in Australia, and had on one occasion been buried in a mine for several hours. This occurred three years ago. A few days after this accident, the first shocks of lightning pains appeared in the right leg; and twelve months before coming under my care, ataxy of gait, with other characteristic symptoms, began to be perceptible. The patient denied any syphilitic infection.

In other cases we find accidents mentioned, together with syphilis, a wild life, and other causes :—

Case 20.—An officer, aged forty-three, married, but childless, consulted me in October, 1880. He had been masturbating as a boy, and led a very wild life later on. In 1864 he had a chancre, and syphilitic manifestations had been breaking out from time to time ever since. He had lately suffered from an obstinate syphilitic ulceration in the legs. He had also had a severe accident while hunting. In 1877, *i.e.*, thirteen years after the primary affection, he had the first symptoms of tabes, viz., double vision, neuralgic pains in legs and arms, incontinence of urine, and loss of sexual power. On examination he showed Westphal's, Romberg's and Argyll-Robertson's symptoms; he stated that he passed his water habitually into bed, and that, although he was completely impotent, he still had sexual desire. The walk was ataxic, but he could still walk for ten or fifteen miles at a time. There was no affection of sensibility in any part of his body.

The following case shows a combination of syphilis, excesses in smoking, drinking and sexual indulgence, two several accidents, and habitual exposure to cold :—

Case 21.—A chemist's assistant, aged thirty-six, single, consulted me in November, 1881. He had for years led a very wild life, and exceeded greatly in drinking, smoking, and sexual intercourse. Ten years ago he had a sore which was presently followed by a bubo and roseola, but no other symptoms of infection. Four or five years ago he had, on two several occasions, fallen from his horse. He had also been kept very much exposed to cold in a shop, where no fire was in winter and draughts were incessant. Three years ago, when at a cricket match, he felt that he could not run well, and suddenly staggered and fell down. Ever since that time he has had great difficulty in walking. Two years ago he had double vision, but only for a short time. Shooting pains in the legs then came on, which were, however, not severe ; and at present he complains more of pain in the left shoulder, which is apt to come and go quite suddenly. Going down stairs is most awkward, while he manages to go upstairs tolerably well. Romberg's, Westphal's, and Argyll-Robertson's symptoms are marked. He suffers from constipation of the bowels and incontinence of the urine ; and the sexual power is gone. There is considerable diminution of sensibility in the sphere of the right ulnar nerve ; but none in the left. The muscles of the balls of both thumbs are greatly wasted, and the patient has on this account great difficulty in writing. The interossei are also somewhat wasted. The electric reactions of the wasted muscles are, however, normal. The grasp of the dynamometer was only 60° with the left, and 80° with the right hand.

I saw this patient again in June, 1883, when there were, in addition, symptoms of optic atrophy in the right eye. The difficulty of vision had only become perceptible about a fortnight ago, when " a dark cloud seemed to become settled at the top of the field of vision, and he was unable to distinguish with that eye anything above the eyebrow." There was achromatopsia on the right, but not on the left eye. The right eye showed the peculiar white condition of the nerve, but the left appeared normal by ophthalmoscopic investigation, and also had the proper acuity of vision.

In the next case there was a combination of syphilis and accident, and the symptoms of tabes were rather blurred.

Case 22.—In May, 1872, I was consulted by a surgeon, aged thirty-two, who had five years ago fallen with his horse. Until then he had enjoyed perfect heath. He received no injury except slight bruises, but felt so shaken that he could hardly do anything for about a week, after which he felt much better. He then had to make a hurried journey to Cannes, travelled night and day for a week, and smoked a great many bad cigars. On his return home he felt "very seedy, as if he was going to have a serious illness." From that time a number of secondary syphilitic symptoms made their appearance, although he never to his knowledge had a primary sore. While in France he had had no sexual intercourse whatever. Psoriasis appeared over the greater part of the body; then rupia, ulcerated sore throat, and coppery blotches on various parts. At the same time he had frightful attacks of headache, with giddiness and formication in the lips. He then took perchloride of mercury and potassic iodide, but without benefit; one symptom succeeded another, until he was so weak that he could hardly stir. Then came a dull pain in the lumbar region, which was very much increased by riding or walking. This was followed by difficulty to urinate, pins and needles in the skin over the abdomen, and loss of sensation and motion in the lower extremities, with loss of control over the bladder and rectum. He was then confined to his bed for six weeks, and eventually sent to Aix-la-Chapelle, where, under the care of Dr. Wetzlar, he underwent a course of mercurial inunction and baths.

At present he was obliged to use the catheter habitually for emptying the bladder, and had no control over the rectum whenever the evacuation was loose. Formerly the urine was spasmodically ejected when he walked, and he had no control whatever over the rectum. He had more power in the legs. Formerly, if he tripped ever so lightly, he was sure to fall, while now he rarely tripped, and could generally save himself from falling. In fine weather he could now walk four or five miles ; but if it was damp or thundery, the power of walking would leave him altogether. He had several times had an enlargement of the left testicle, which was generally reduced by taking potassic iodide, but on one occasion an abscess formed and burst. He was now getting very fat and weak, and found it an effort to do anything; but when breathing mountain air he was much better able to exert himself.

A similar combination of accident and syphilis was seen in the following case :—

Case 23.—A bookseller, aged thirty-three, single, consulted me in October, 1876. He had had syphilis about ten years ago. Five years ago he had a fall from a horse, and was pitched down head foremost, and made a somersault in the air. He felt giddy, saw sparks flying before his eyes, but recovered himself almost immediately, and walked home by the side of the horse. About six months afterwards he began to suffer from lightning pains and loss of control over the limbs. He has now lost flesh to such an extent that his legs are mere sticks ; and the same is the case in the arm ; there is no biceps left ; while in the forearm there is more muscle and more power. The gait is of the purely ataxic type ; the patient cannot walk at all, except when supported, and is worse in the dark. There is loss of control over the bladder and rectum, and the sexual power and desire are gone. He suffers also very much from indigestion, and has a very feeble pulse. "When the liver is out of order, the pain becomes much worse" (no doubt an instance of the coincidence of "gastric crises" with bouts of lightning pain).

eal excesses have already been mentioned in several which I have just shortly related. Duchenne has seen a case in which "frantic masturbation" appeared to be the cause. Sexual excesses generally coincide with excesses in drinking, smoking, and the acquisition of syphilis, and it is therefore difficult to apportion the exact degree of influence which the former agent may possess. I have not seen a single case in which venereal excesses alone could be looked upon as the cause. In the female sex, tabes is chiefly found in prostitutes; and in them the three agents of syphilis, excesses, and drink probably rank in importance in the order in which they have just been stated in the production of the malady. Exposure to cold may in some instances be added to it. Lancereaux[1] has noticed that tabes occurs amongst girls in Paris who work the sewing-machine with the foot. These women are extremely hard worked, as many of them have to continue at their work from six a.m. until midnight. The constant movement of the foot appears to excite the sexual organs, which become so hot and congested that the women are obliged to wash the parts frequently with cold water. It is assumed that the excessive functional excitement starting in the sexual organs provokes through the centripetal nerves irritative reaction in the parts whose function it is to receive those impressions, and that it is more the prolonged and injurious action of a more or less abnormal sexual excitement on the spinal cord than syphilis which produces tabes. I am unable to agree with this opinion. I have seen in hospital practice cases of tabes in London working women who had to use the sewing-machine which is worked with the foot, and have inquired amongst them whether this has the effect on the sexual organs mentioned by Lancereaux. They did not seem to understand the bearing of my inquiry, although they happened to be very intelligent persons; but attributed their illness to having worked in damp workshops and amongst generally un-healthy surroundings. None of them had apparently had syphilis. The French working girls are probably sexually more excitable than the English ; a similar difference being seen in the development and aspects of hysteria in the two countries.

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[1] "Transactions of the International Medical Congress," 1881, vol. ii., p. 42.

The influence of *drink* alone does not seem to be great. A French army surgeon (Case 24), who consulted me in December, 1882, for a very painful form of tabes, with severe laryngeal crises, mentioned in his history masturbation early in life ; syphilis, sexual excesses, and *absynthe-drinking* later on. The influence of *drink in the parent* was the only cause which I could discover in the case of a Swedish gentleman (Case 25), who was sent to me by Dr. Allan, of Hyde Park Terrace, in January last. He had led an exemplary life, had never had anything to do with women, not met with accidents, nor been subject to exposure or unfavourable influences of any kind. His father, however, had been an inveterate drunkard, and had died in a fit of drunkenness at thirty-nine years of age. It is therefore not at all improbable that this patient may have been conceived while his father was in a state of intoxication—a circumstance which is known to give rise to epilepsy, paralysis, idiocy, and insanity in the offspring, and is probably instrumental in inducing the form of tabes which is known as Friedreich's disease.

The influence of *tobacco-smoking* is probably not great ; excessive smoking, however, was mentioned to me as the cause of his illness by a merchant (Case 26), aged thirty-one, married, and father of three children, who consulted me in March, 1874. He said that he had for years smoked all day long without interruption, and habitually consumed as much as two ounces of birds-eye per diem. He had never had syphilis, nor been subject to any other unfavourable influence. His present illness dated from three years ago, when his legs became very weak and numb. He now feels " as if he had no legs, but only two sticks." He has the characteristic difficulty in standing, the ataxic gait, loss of sexual power, and of control over the bladder and bowels.

The *acute infectious diseases* appear to have but little influence in producing tabes. I have never seen a case occurring subsequently to typhoid fever, small-pox, erysipelas, measles, etc. ; and only one after jungle fever.

Case 27.—This was the case of an officer, aged thirty-five, single, who was sent to me by Dr. Henry Savage, in May, 1865 ; he had been fourteen years in India, and enjoyed good health until about twelve months ago, when he was seized by jungle fever, which proved most intractable ; and he never recovered his health thoroughly afterwards. He suffered much from indigestion and nervousness, but more particularly from difficulty in walking. I found the gait typically ataxic. He was apt to lose his balance, and had frequently fallen down lately ; felt quite helpless in the dark, was very nervous on going down stairs, and had a peculiar feeling in walking, as if the ground rose with him, and he were walking on elastic springs. The right pupil was larger than the left, but there were no ophthalmoscopic signs of optic atrophy. He had had no syphilis, and his illness could only be attributed to the effects of the fever. There was a slight but perceptible swelling of the spleen.

Cases of acute tabes have been seen in connection with lepra and pellagra. After ague, Kahler and Pick have noticed absence of the knee-jerk, which eventually returned when the patient's health was restored. Poisoning with lead, arsenic, barium, and other metals seems also occasionally to lead to acute tabes ; but this part of the subject is as yet hardly explored.

In some few patients, finally, we do not find any other cause except the *neurotic constitution* as inherited from parents who have been subject to epilepsy, paralysis, insanity, hysteria, chorea, megrim, malformation of the skull, or simply to excessive excitability of the nervous system, as shown by habitually violent manners, outbursts of passion, an odd or eccentric behaviour, etc. Eulenburg[1] mentions the case of a patient in whose family there had been direct transmission of tabes from father to son for four generations. Trousseau[2] has known a family in whom one member was affected with tabes while others were subject to monomania, hypochondriasis, and spermatorrhœa; another patient with tabes had an aunt and an uncle who were both insane, a brother who had also tabes, and another brother who had hemiplegia; and a third patient, who had tabes, was the son of a father who had committed suicide, and had himself two sons, one of whom, although not insane, was in the habit of uttering piercing shrieks all day long, "impelled thereto by an irresistible force," while the second had a singular form of muscular "tic." Vidal, quoted by Topinard,[3] reports the case of a man with tabes, whose sister died in a madhouse, one of whose daughters died in a fit, while another suffered from incontinence of the urine; the patient himself was affected by congenital nystagmus. In a patient of Gubler's, the maternal grandmother and a paternal uncle had died of hemiplegia, while his mother had long been hysterical, but was then alive and well, at eighty years of age.

The influence of the neurotic constitution, however, is more particularly seen in that peculiar form of tabes which is known as Friedreich's disease (p. 65), or hereditary ataxy. Friedreich's own observations, which have since been supplemented by those of others equally striking, extended over three sets of cases, viz., first a brother and sister, whose father had been a notorious drunkard;

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[1] *Loc. cit.*, p. 458.

[2] "Clinique Médicale de l'Hôtel Dieu de Paris," vol. ii., p. 610. Cinquième edition. Paris, 1877.

[3] "De l'Ataxie Locomotrice," p. 444. Paris, 1864.

second, three sisters, whose parents were industrious and healthy; and, finally, three sisters and one brother, whose father seems to have been a somewhat remarkable character, as he combined the functions of tailor, barber, and musician, drank heavily, led a fearfully immoral life, and eventually died of consumption. The wife of this man was bodily healthy, but unusually stupid; she mentioned, however, as a fact that all the four children had been conceived while the father was in a state of intoxication. I have already (p. 110) spoken of the case of a patient now under my observation, in which this appeared to be the only cause of the tabes which could be ascertained.

Carré [1] has given the history of a family in which the grandmother, the mother, and all her relations, who numbered eight, as well as seven children and one cousin, in all eighteen, were affected by tabes. Of the seven children, three were dead; one was deaf, and crawled about on all fours; while the cousin was blind. In 1872, Dr. Carpenter, of Croydon, showed to the Medical Society of London two girls suffering from what he called "muscular anæsthesia," but what was really Friedreich's disease; and later on a third member of the same family became affected.

Dreschfeld [2] has described a family where five, viz., three brothers and two sisters, out of fifteen, were tabid; and Gowers,[3] one in which five out of eight had the same disease, viz., one girl and four brothers. None of these latter patients had had syphilis or inherited syphilis; but on the father's side there was a long history of insanity, while the mother had suffered from chorea; and the disease commenced in all the patients between the ages of eighteen and twenty-one.

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[1] "De l'Ataxie Locomotrice Progressive." Thèse de Paris, 1863.

[2] "Liverpool and Manchester Medical Reports," 1876, vol. iv. p. 93.

[3] "Transactions of the Clinical Society of London," 1881, vol. xiv. p. 1.

Wälle,[1] of Wattwyl, describes two cases of Friedreich's disease, which are remarkable from having begun at an unusually early age. Both patients were males, and came of a family of seventeen children, of whom seven died in infancy. The father had been healthy, but the mother had been temporarily insane; and there had been no other nervous diseases in the family. The eldest of the two brothers, now twenty years of age, was in his *seventh year* taken with difficulty in walking, followed presently by awkwardness in the use of the upper extremities. He had sometimes pain in the knees and legs, although not of the lightning or terebrating character, and occasionally headache and vomiting. At present there was curvature of the spine, with the feet in equine position, and dorsal flexion of the big toes; the muscles were not wasted, and showed considerable power. The patient was awkward in taking hold of objects, and, on closing the eyes, could not put a tumbler to his mouth. There was numbness in the feet, and sensibility was altogether somewhat reduced. All the deep reflexes were absent. There was slight nystagmus, and the speech somewhat drawling. In walking, the ataxy was so severe that the patient could not attempt it without support.

The second patient was twelve years old, and had had difficulty in walking since his *fourth year*. There was also lateral curvature and commencing equine position of feet. He walked like one who is drunk, and the deep reflexes were absent. Sensibility, however, and the use of the upper extremities were not interfered with.

Friedreich thought that the female sex was more liable to the affection than the male, as seven of his cases occurred in girls and only two in boys; but, out of forty-six cases which I have collected, thirty occurred in boys and only sixteen in girls, showing a decided preponderance of the male sex. As regards age, the malady commences in a good many cases about the time of the development of puberty, that is, between twelve and seventeen years; and in connection with this it is useful to remember that Rokitansky has found great tendency to venous hyperæmia in the spinal canal during that period of life. The general tendency of Friedreich's disease to appear at an early time of life is one of the features distinguishing it from ordinary tabes, which usually becomes developed between thirty and fifty years of age.

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[1] "Correspondenzblatt für Schweizer Aerzte," 1883, p. 145.

While, therefore, in Friedreich's disease, and also in some few cases of ordinary tabes, a neurotic predisposition seems undoubtedly to exist, I have come to the conclusion that in by far the largest majority *of cases of ordinary tabes the fault lies more with the individual patient than with the stock from which he came.* Hysteria in the mother I have found in several cases, while of other forms of nervous disease in parents I have seen singularly little. Vulpian states that a special predisposition must necessarily exist for the development of tabes, even where there has been syphilis; for why, he asks, should tabes be so rare when syphilis is so frequent? To this question I may answer, first, that tabes is much more frequent than is generally believed; and, secondly, that many persons who have acquired syphilis are subjected to a long course of specific treatment, and therefore escape the tertiary affections which might otherwise have been in store for them. On the whole, therefore, there is only a slight influence of heredity in tabes; on the other hand, the mode of life adopted by the individual plays, no doubt, a considerable part in the production of the disease. Persons who have had syphilis will be more likely to acquire tabes if they commit afterwards excesses in sexual indulgence, the pleasures of the table, drinking and smoking, and lead an irregular and exciting kind of life.

Fournier finds most victims of tabes amongst the votaries of pleasure in large cities. Yet syphilis, drink, and venereal excesses are quite as much at work in small country towns as in Paris and London, and I doubt whether tabes is really more frequent in the "great centres of civilisation." I have seen a considerable number of men who had emptied to the last dregs the cup of what is called "pleasure," and who, having by a lucky chance steered clear of syphilis, had not acquired tabes. Excesses without syphilis appear to lead in general more to cerebral than to spinal disease.

Whether any particular profession is more exposed to tabes than another is difficult to ascertain. In hospital practice my patients have been firemen, boatmen, cabmen, coachmen, policemen, engine-drivers, workers in drainage, railway guards, and navvies ; and amongst women those who had to work hard and long in damp places, more especially upholsteresses, waistcoat-makers, laundresses, etc. On the whole, however, tabes appears to be more frequent in the higher than in the lower strata of society ; and this is possibly due to the greater mental efforts required of the former, combined with anxiety and depressing emotions of various kinds which affect the rich more than the poor, and which tend to lower the power of resistance of the nervous centres to injurious influences.

Whether acute or subacute muscular *rheumatism* has any influence in the production of tabes, as has been stated, seems very doubtful. Most patients call the lightning-pains rheumatic, and come with an apparent history of rheumatism, which, however, does not bear critical investigation. The same may be said of our old friend, the "*suppression of habitual perspiration.*" This is rather more likely to be owing to, than productive of, the disease. A patient who was sent to me by Mr. Bickersteth, of Liverpool, in June, 1882 (Case 27), ascribed the outbreak of the malady to a bad attack of *diarrhœa*, which he had had while staying at Madeira ; it was then that he first noticed unsteadiness in walking, which the doctor there thought to be owing to the relaxing climate. On inquiry, I found that he had had syphilis very badly fifteen years ago. He then suffered from fearful ulcerations of the skin and subjacent tissues, buboes which would not heal, etc., for about fifteen months after the primary sore, and he acknowledged having been insufficiently treated "from want of time"! He has now occasionally crops of copper-coloured spots appearing on his skin; on the whole, however, the "active" manifestations of the disease have ceased for some years past. At present he shows absence of knee-jerk in both legs; has a difficulty in holding and expelling the urine, looseness of the bowels, diminished sexual desire and power, Romberg's symptom, ataxic gait, and difficulty in going downstairs. In the upper extremities the muscular force is considerable, as he squeezed the dynamometer to 160°, yet the fingers were numb. He had never suffered from lightning-pains or constriction round the waist. While, therefore, the disease was no doubt owing to syphilis, yet the attack of diarrhœa may have aggravated the already existing malady.

We are on much firmer ground in discussing the influence of *age* and *sex* in the production of tabes.

With regard to the age at which the disease is most likely to break out, the statements of several observers are devoid of value, as they have only given the ages at which the patients came under care, and not those at which the first symptoms of the malady appeared. Thus Topinard,[1] who has tabulated 104 cases, gives the following list, which is also objectionable by its arrangement, as the same numbers are made to appear at the beginning and the end of the period :—

From 26 to 30 years of age 13 cases.
" 30 " 35 " 11 "

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[1] "De l'ataxie locomotrice," p. 359. Paris, 1864.

From 35 to 40 years of age 20 cases.
„ 40 „ 45 „ 20 „
„ 45 „ 50 „ 20 „
„ 50 „ 55 „ 10 „
„ 55 „ 60 „ 6 „
„ 60 „ 65 „ 3 „
At 75 years in 1 case.

This list only applies to the fully-developed malady, when the patients are in the second period. Erb,[1] on the other hand, finds the beginning of the malady—

Between 11 and 20 years in 3 cases.
„ 21 „ 30 „ 13 „
„ 31 „ 40 „ 31 „
„ 41 „ 50 „ 18 „
„ 51 „ 60 „ 3 „

In fifty-two cases in which I have been able to ascertain as nearly as possible the date of the outbreak of the disease, it occurred—

At 9 years in 1 case.
Between 20 and 29 years in 9 cases.
„ 30 „ 39 „ 18 „
„ 40 „ 49 „ 13 „
„ 50 „ 59 „ 10 „
At 60 years in 1 case.

My youngest patient was a girl, aged nine (Case 28), who was sent to me by Dr. Horace Dobell, in July, 1881. She had been staying at the seaside, and been paddling a good deal in the cold water with her naked feet, which was the only cause that could be ascertained. After a time, she experienced lightning pains in different parts of the body, which were short, sharp, and agonizing, and caused her to scream whenever a shock passed through her. When she came to see me, these shocks occurred chiefly in the left knee, where a stabbing pain was felt every two or three minutes. The knee-jerk was absent in both sides. There were no other symptoms ; and she did not appear to have ever been subject to symptoms of inherited syphilis. The pain resisted the use of nitrate of silver, in doses of one-eighth part of a grain, and the application of the constant current ; but yielded completely to the use of ergot, in ten-minim doses of the liquid extract, thrice daily. I have not seen the little patient since, and am therefore unable to say whether the patellar reflex has returned.

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[1] "Krankheiten des Rückenmarks" (in Ziemssen), vol. ii., p. 131 Leipzig, 1878.

The next youngest patient with tabes (Case 29) whom I have seen was an omnibus-conductor, aged twenty-two, who came under my care at the hospital in February, 1878. He had had small-pox ten years ago, and a primary sore when seventeen years of age. He appeared to have had no secondary symptoms, but had an attack of hemiplegia of the left side two and a-half years ago, being then in his twentieth year. He recovered from this completely in three weeks. In June, 1877, he had an attack of double vision, which was treated at Moorfields. A week before he came to the hospital he suddenly felt pain at the back of the head and down the neck, and a curious sensation of numbness in his face, which soon afterwards was drawn to the side. When I examined the patient, there were all the symptoms of paralysis of the left portio dura in the first portion of the Fallopian canal, together with paralysis of the left rectus externus. *The knee-jerk was absent in both sides.* There could be no doubt that it was a case of commencing tabes ; but it was difficult to say when this affection commenced, and whether it was already present when the patient had the stroke of paralysis at nineteen years of age, or whether the attack of double vision at twenty-one was the first symptom of it. An examination of the knee-jerk alone at the earlier period of his illness could have settled this question.

Another case in a very young man was that of a lieutenant in the Indian army (Case 30), who was sent to me by Mr. Erichsen in April, 1872. He was then twenty-three years of age, and had contracted syphilis three years ago. He had had an indurated chancre and bubo, and had subsequently suffered from severe ulceration in the region of the left shoulder, and from iritis. Symptoms of tabes appeared about twelve months ago, that is, at twenty-two years of age, and were more marked in the left than in the right side of the body. He was much troubled with numbness, particularly in the left foot; had difficulty in standing on the left foot, while he could stand tolerably well on the right; staggered when he was requested to close his eyes; and had much difficulty in walking slowly, while he could walk fast better. He then, however, had to manœuvre considerably, and showed the characteristic gait of ataxy in the most unmistakeable manner.

From the table which I have just given, it appears that tabes is most prone to break out between thirty and fifty years of age. The ages of the same patients at the time they came under my care were as follows:—

At 9 years in 1 case.
From 20 to 29 years in 3 cases.
„ 30 „ 39 „ 12 „
„ 40 „ 49 „ 21 „
„ 50 „ 59 „ 11 „
„ 60 „ 69 „ 3 „
At 72 years in 1 case.

Tabes is, therefore, in general a disease of the prime of life.

The following table is given by Eulenburg:—

The disease appeared at 9 years in 1 case.
„ „ up to 20 „ 2 cases.
„ „ „ 30 „ 37 „

The disease appeared up to 40 years in 46 cases.
" " " 50 " 48 "
" " " 60 " 5 "
Above 60 " 0 "

Leubuscher[1] has reported a case where a boy, aged three and a half, was affected, and no neurotic tendency could be traced in the family; Eulenburg, one in a girl aged nine; and Bradbury,[2] in a young man aged nineteen. On the other hand, Trousseau describes the case of a patient at eighty years of age, where the muscular force appeared to be quite intact. It is, however, not quite clear that this really was a case of tabes.

The oldest patient with unmistakeable tabes whom I have seen was a gentleman aged seventy-two (Case 31), who consulted me in June, 1881, and who had had syphilis thirty years ago. The first symptoms of tabes appeared in him three years ago. In the same month, I saw another gentleman, aged sixty-six, a widower (Case 32), who had had syphilis twenty-six years ago. He had remained well for sixteen years, and it was only then that the first symptoms of tabes presented themselves. At present he shows all the usual symptoms of the disease confined to the lower dorsal and upper lumbar portion of the cord. Such cases, however, are exceptional.

With regard to *sex*, all observers are agreed that the male is much more liable to tabes than the female. I have never seen this disease in a lady, but a good many cases of it amongst the out-patients of the hospital. These were chiefly women who had to work in damp and unwholesome shops, and some of them had had syphilis.

Möbius[3] has lately described five cases of tabes in women, four of whom had syphilitic husbands, and who had

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[1] "Berliner klinische Wochenschrift," 1882, No. 39.

[2] "British Medical Journal," 1871, No. 565.

[3] "Centralblatt für Nervenheilkunde," p. 193, May 1, 1884.

had frequent miscarriages, while all other causes of the disease, such as exposure to cold, over-exertion, alcoholism, injury, etc., were wanting. These cases are particularly important, because in men there are generally other injurious influences at work, as for instance in Cases 20 and 21 (p. 105) where a variety of causes might have led to the outbreak of the disease. Singularly enough, there were in none of Möbius' female cases any of the ordinary signs of secondary syphilis; yet their history leaves no doubt on the mind that in these tabes was really of a specific character. If we consider how difficult it frequently is to ascertain the existence of syphilis in women, and how eager they generally are to deny that they ever had such a disease, it will be acknowledged that some positive evidence is of much more importance than numerous negative statements. Moreover it is well known that the immediate consequences of infection are often in women so exceedingly obscure that they are themselves unaware of being infected; and the fact that in men often just the apparently mild forms of the disease appear to lead to tabes (p. 84), no doubt also applies to the female sex.

Posterior sclerosis may finally be a secondary disease, becoming developed consequently upon other affections of the cord, such as myelitis from compression, Pott's disease, chronic meningitis or meningo-myelitis, secondary sclerosis of the lateral columns, etc. Such cases, however, do not offer the peculiar type of the primary disease; the symptoms are often indistinct, and it is difficult to make an accurate diagnosis.

* * * * *

The causes of *spastic spinal paralysis* are analogous to those of tabes; yet I have found certain differences which appear to me worthy of note. Lathyrism, corresponding to ergotism, has already been mentioned (p. 71). But in that form of the disease which occurs habitually in practice, some points have struck me forcibly: viz., first, that the influence of the *neurotic constitution* is much more marked in the production of this disease than in tabes; second, that the occurrence of spastic paralysis is apparently not so much influenced by *sex and age* as tabes; and third, that a *syphilitic* history is much more rarely obtained in this affection than in tabes.

The influence of the neurotic constitution was strongly marked in a single gentleman (Case 33), aged sixty-two, who first consulted me in January, 1878. He was one one of twelve children, whose truly tragic life-history is as follows:—The eldest sister died of consumption at twenty-three years of age; the eldest brother committed suicide at thirty-one. The next sister died in infancy; another, who is her twin-sister, is alive, and has for many years past suffered from an intractable form of epilepsy. The next is the patient himself, who has for some years past been subject to spastic spinal paralysis. The next brother died suddenly, aged twenty. The next sister is alive, and highly nervous; the next one died at fifty, from heart-disease. After that come two twin-brothers, another brother and a sister, all of whom were habitual drunkards, and died of the effects of drink.

In another patient (Case 34), a young lady, aged twenty-two, for whom I was consulted in May, 1878, the parents had been first cousins; the mother was highly hysterical, and a maternal aunt was insane. In a number of other patients hysteria of the mothers was ascertained; and in most of these cases no particular exciting cause of the outbreak of the disease could be elicited.

As far as sex is concerned, the female seems to be quite as liable to it as the male, as amongst forty-nine cases of which I have notes, I found twenty-four males and twenty-five females. The ages at which the disease first appeared were as follows:—

Up to 10 years... ...	5 cases.
From 11 to 20 „	8 „
„ 21 to 30 „	9 „
„ 31 to 40 „	8 „
„ 41 to 50 „	10 „
„ 51 to 60 „	8 „
After 60 „	1 „
	49

If this list is compared with the one of the ages at which tabes has appeared (p. 120), a considerable difference will be noticed.

Syphilis has already been mentioned (p. 53) as having evidently caused the affection in Minkowski's case ; and Westphal has published a similar one. The following are a few cases taken from my own note-book :—

Case 35.—A sailor, aged twenty-eight, married, and father of two children, was admitted into the hospital under my care in April, 1882. He had had small-pox when eight years of age, and had contracted syphilis four years ago ; but he attributes his present affection to having got wet at sea. Three months ago he felt that he was gradually losing the power over both arms and legs ; and he is now so feeble that he cannot walk at all. There is great exaggeration of the deep reflexes in all four extremities ; no wasting nor unusual electrical reactions ; there is incontinence of the urine, and habitual constipation ; and there never has been any pain or other affection of sensibility.

Case 36.—A surgeon, who had lived a good deal in the East and on board ship, consulted me in September, 1881. He had had syphilis first in 1861, and then again very badly in 1865. In 1866 he was taken ill at Calcutta with fever, but nevertheless went off in a ship, taking charge of coolies for Trinidad. He afterwards came to England, and was laid up with jaundice ; he had retention of the urine and convulsions in the left leg, and became so feeble that he was weeks before he could crawl from his bed to the sofa on all fours. He now presented spastic gait, great increase of all the tendon reflexes, and no affection of sensibility.

Case 37.—In July, 1883, I was consulted by an American gentleman, aged forty-five, single, who had masturbated as a boy, and had syphilis twenty years ago. For some years past he had felt his sexual desire and power gradually lessening ; and at present there was total anaphrodisia and impotency. In addition to this, the patient suffered from constipation and frequent attacks of "congestion of the liver." He also complained of general debility and not being able to walk well. The gait was that of incipient lateral sclerosis ; and the deep reflexes were considerably exaggerated in all four extremities.

A not infrequent cause of this form of sclerosis appears to be *external injury*. A fall from a horse was mentioned to me as the only possible cause of his illness by a merchant, a native of Barbadoes, aged fifty (Case 38), married and childless, who consulted me in June, 1883, and who had for about twelve months past, suffered from gradually increasing debility in walking (spastic gait), and had become much emaciated in his lower extremities. His arms and hands were likewise much weaker than they had been before. The tendon reflexes in all four extremities were greatly exaggerated, with spinal type. There was no affection of sensibility, and the bladder, bowels, and sexual organs appeared to be in their normal condition.

A similar case was that of a single lady, aged sixty-three (Case 39), who consulted me in January, 1882. Ten years ago, when visiting a picture gallery in Rome, she accidentally put her foot into a hole in the floor and fell, severely jarring her spine. Ever since then she has not been able to walk well, and has constantly, although very gradually, got worse. On examining the legs, the muscles appeared well nourished, and responded readily to faradisation. There was no anæsthesia, but great exaggeration of all the deep reflexes. The patient could only walk with support on both sides, and showed the characteristic gait of spastic paralysis.

Another single lady, aged fifty-one (Case 40), consulted me in the same week. She had always been in good health until eight years ago, when the horses of the carriage in which she was driving took fright and upset it. She was thrown out, and hurt the lumbar portion of the spine. There was no fracture or dislocation, but bruises and severe pain; and the patient has never been able to walk properly since. There is now extreme exaggeration of all the tendon reflexes in both lower extremities, and all the other symptoms of spastic paraplegia. More recently the right arm has also become very weak. With the right hand she squeezes the dynamometer only to 40°, while with the left she has a grasp of 100°. She complains of pain in the right arm, about the region of the insertion of the deltoid muscle. She has difficulty in lifting the right arm, is unable to do her hair, or to dress herself without assistance. The tendon reflexes of the right arm and hand are exaggerated, and present a striking contrast to the left upper limb, where there is no such exaggeration. Otherwise she is in good health. Her catamenia left her without any trouble twelve months ago; and she has no difficulty with the bladder and rectum.

Spastic paralysis sometimes appears after *acute diseases:* I have seen it after scarlet fever and typhoid fever. One of my patients, a lady, aged thirty, attributed her malady to *over-exertion* in nursing a brother day and night through
us illness which required constant attendance, and
ially incessant lifting and shifting of position.
a clerk, aged thirty (Case 41), the symptoms

came on after violent catheterisation. This patient had had gonorrhœa, which left a stricture, and was subject to occasional attacks of retention of urine. In one of these attacks a false passage was made with the instrument, after which severe symptoms of urethritis and cystitis became developed. When the patient recovered from this, he found that he had difficulty in walking; and when I examined him in February, 1883, the symptoms of spastic paralysis were well marked.

The causes of *insular sclerosis* are as yet not well known. In a case of this kind, which I saw with Dr. Philpot, of Croydon, in October, 1882, the patient, an auctioneer, aged forty-one (Case 42), had been twice married, had had a chancre and a bubo at eighteen years of age, but apparently no secondary symptoms. He confessed to considerable sexual excesses. About three and a half years ago he began to find his speech becoming affected; articulation was drawling and indistinct. This had got much worse lately, but the patient had full command over his words; and the tongue was neither tremulous nor wasted. The head was bent forward on the chest, from paresis of the trapezius and other muscles of the neck; but with an effort the patient was able to throw the head backwards, and also to move it from one side to the other, without much tremor. There was, however, well marked sclerotic tremor in the left side of the body on moving the limbs, but only very slightly so during rest. He squeezed the dynamometer with the right hand to 160°, and with the left to 120°; and although there was, therefore, a very good degree of muscular force, yet this appeared to be of very little use to the patient. It is true that he could write, but very slowly and awkwardly. He could button his trousers, but with great difficulty; he was unable to cut his meat, and had to feed himself with a spoon. The sclerotic tremor of the left arm was much increased when he attempted to speak. The backward motion of both hands, as when he wanted to use the hair-brush, was very much interfered with. The spine felt lumpy, clogged, and weak. He had often shooting pains through the back, with shivering. He could not sit straight in a chair, and preferred lying to sitting, and running to walking. He could still walk about three miles a day, but perspired profusely after having walked for about a mile. All the deep reflexes were exaggerated ; but there was no affection of sensibility, and no symptoms of syphilis in the skin, mucous membranes, and bones.

The neurotic constitution was very obvious in a married lady, aged thirty-five, and mother of two children (Case 43), whom Mr. Maclaren requested me to see, in April, 1883. She had for five years past suffered from a gradually increasing loss of power in the lower extremities. *Her father and mother had died of apoplexy, and a sister had suffered from infantile paralysis.* The disease commenced without any appreciable cause, and the patient had, throughout all these years, never had an ache or pain. She was now confined to her couch, but could sit up on a comfortable chair for some time. The legs were utterly useless, and almost entirely paralysed ; she could, however, push the left leg down when it was drawn up. Both legs were much emaciated, and the faradic excitability of the nerves and muscles was diminished, while the voltaic response appeared normal. The knee-jerk was much increased in both sides, but there was no ankle-clonus. Sensibility was perfectly normal. The upper extremities were likewise very useless and wasted. She could not wash her hands, write, dress, or feed herself. The dynamometer showed 25° with the right, and 30° with the left hand. There was nystagmus and a slight degree of excavation of the optic nerves, without, however, any corresponding loss of sight.

Insular sclerosis is not uncommon in children, and has been observed at so early an age as fourteen months, but is more common in them between three and four years of age. In children a blow or a fall is often supposed to be the cause, although no very definite relationship has as yet been established between accidents and insular sclerosis. On the other hand, it is certain that the disease is apt to appear after scarlet fever, jaundice, typhoid fever, cholera, and small-pox. Whether these morbid poisons have a tendency to invade directly certain portions of the nervous system, or whether the latter is affected secondarily, in consequence of certain systemic changes brought about by the action of these poisons, is as yet unsettled. Insular sclerosis has also been observed in gouty subjects ; and it appears to be occasionally hereditary.

Only little is known at present about the causes of *amyotrophic lateral sclerosis.* It generally occurs in persons between thirty and fifty years of age ; but Seeligmüller has seen four children of a family affected with it, whose ages were between one and eleven years. Syphilis and exposure to cold have appeared to me the most probable causes of this variety of sclerosis.

CHAPTER VI.

SYMPTOMS OF TABES SPINALIS.

THAT the course of tabes spinalis shows several distinct stages was first recognised by Duchenne,[1] who divided the disease into three different periods. The first was, according to him, characterised by the occurrence of palsies of one or several of the motor nerves of the eye, or their branches, of amblyopia and amaurosis, and of lightning-pains; the second, by symptoms of motor incoordination in the lower and sometimes in the upper extremities, to which, soon afterwards or at once, were added anæsthesia of the muscles, joints, bones, and the skin; while in the third period the malady became generalised. These three stages have subsequently been called (1) the premonitory, prodromial, or pre-ataxic stage, (2) the ataxic, and (3) the paralytic or terminal stage; and they are sufficiently well marked in a large number of cases to warrant us in adhering to this division. In other cases only two stages can be distinguished, viz., a pre-ataxic and an ataxic one, as the patients do not live long enough to reach the third or paralytic stage. The first stage should not be called "premonitory" or "prodromial," because the peculiar lesion of tabes is already established in the beginning of the malady, which latter is at no period functional in character. The *pathological difference between the several stages is therefore only one of degree*, inasmuch as in the first stage a small number, in the second a much larger number, and in the last the totality of the nerve-tubes of the posterior columns are diseased or destroyed.

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[1] "Archives Générales de Médecine," December, 1858; Jan., Feb., and March, 1859.

From this it naturally follows that no hard and fast line can be drawn between the several periods of tabes ; and this view, which is suggested by the pathological anatomy of the complaint, is fully corroborated by the clinical symptoms. It is, therefore, in many cases impossible to say whether a patient is in the first or second, or in the second or third stage of the disease. The ataxy of movement, as well as of standing, is often so slight that it can only be discovered by careful examination ; and it would be inexpedient to say that a patient, who may still be able to walk eight or ten miles at a stretch without any difficulty, is in the second stage of locomotor ataxy. On the other hand, the ataxy of gait may be so severe that, although the patient may still be able to move his limbs, he is more helpless than another patient who may have been actually paralysed through a stroke of cerebral hæmorrhage. The stages or periods of the disease should, therefore, be looked upon as only approximatively true to nature.

The clinical signs of tabes have gradually become more and more numerous as greater attention has been directed to this disease ; and the descriptions given by Romberg and Duchenne, excellent as they are, will now be found to contain only a fraction of the symptoms which are now habitually observed in patients of this kind. In order therefore, to render this part of my subject as clear as possible, I shall begin with and lay chief stress upon the most important symptoms, and those which are of the greatest value in enabling us to make an early diagnosis of the disease. These are—

1. Loss of the knee-jerk (Westphal's symptom);
2. Lightning pains;
3. Reflectory rigidity of the pupil (Argyll-Robertson's symptom).

If these three symptoms are found together, the diagnosis of tabes may be considered certain.

1. *Loss of the knee-jerk.*—This symptom, which was discovered by Westphal in 1875, is, without exception, diagnostically the most important one in the earliest stages of tabes. In health, a more or less smart tap on the patellar tendon with a finger or the ulnar side of the hand, the ear-piece of a stethoscope, or, better still, with a percussion hammer, causes the leg to be jerked forward more or less freely. The phenomenon may be obtained when the patient is sitting on a chair, and crosses one leg over the other; but where the legs are short and stout, it is better to let him sit on the edge of a table, with his legs hanging down. Where the patient is examined in bed, the limb should be raised by the observer passing his left hand underneath the thigh, just above the knee, and giving the tap with the right hand. In general, it is not necessary for the patient to be undressed; but where the result is doubtful, it is always best to strike the bare skin over the tendon; and it may be useful, more especially where the patient appears nervous and excitable, to have his eyes bandaged, so that any interference on his part with the production of the phenomenon may be prevented.

The controversy about the exact nature of the phenomenon, which commenced soon after its discovery, is still progressing, and likely to continue for some time to come. The highly complex character of an apparently simple sign affords considerable opportunities for the exercise of physiological ingenuity; and it is satisfactory to find that experimental physiologists, who owe the knowledge of this physiological sign to a doctor, should have taken up the study of it with so much zeal, although, perhaps, somewhat late in the day, considering that it is within their own special province.

Westphal and others consider the knee-jerk to be owing to a direct stimulation of the muscular substance, while

Erb and others maintain that it is produced by reflex action. There is a considerable amount of evidence in favour of either theory, into which this is not the place fully to enter. Suffice it to say that, according to Westphal, the essential conditions for the production of the knee-jerk are, that there should be a proper muscular tone, a certain degree of tension of the muscle, and the possibility of the tendon vibrating ; and that there is no need for assuming reflex action by centripetal nerves of tendons and muscles, but that the contraction of the muscle is caused by the specific stimulus of the vibrating tendon. Erb, on the other hand, has pointed to the discovery of nerves in tendons, which was made by Sachs[1] about the same time that the knee-jerk first attracted attention. These nerves are situated at the point where the muscle and tendon touch one another ; they are excited when the tendon is struck, and conduct the stimulation to the spinal cord, the tendon being an elastic medium which only wants a certain degree of tension for transmitting the concussion. In order that the knee-jerk should be producedit is necessary that the reflex path between the tendon, the spinal cord, and the rectus femoris should be in a state of integrity; and where this is interrupted, as in tabes, by disease of the posterior roots and columns, in infantile paralysis by disease of the anterior grey matter, and in neuritis by disease of the peripheral nerves, the knee-jerk is found to be absent. In spite of a considerable number of physiological researches undertaken by such observers as Tschirjew, Gowers, Berger, Brissaud, Eulenburg, and many others, with the view to arrive at a definite conclusion, both Westphal and Erb have until now adhered to the position originally taken up by them ; yet signs are not wanting that the reflex theory is destined eventually to gain the day. The principal argument of the opponents of the reflex theory is, that the time in which the knee-jerk is produced is too short for a true reflectory process, inasmuch as the period required for a stimulus to travel from the patellar tendon to the cord, and from the cord back again to the quadriceps femoris, would amount at least to one-fifteenth part of a second; while as a matter of fact much less time intervenes between the two actions, viz., from the twenty-fifth to the thirtieth part of a second. This view, to which more especially Gowers[1] attaches considerable importance, is, however, controverted by Rosenheim,[2] who has made the latest experiments on this subject in the physiological laboratory of the University of Berlin, and who has found that in men, as well as in rabbits, the least duration of latency is 0·025 of a second, but that it is often much more, and that this is quite compatible with the reflex nature of the phenomenon. He objects to the mode of experimentation adopted by Gowers and Eulenburg as containing sources of error, and has adopted an entirely different mode of research, which has led to different results. He finds that the portion of tendon which is struck is of importance, and that the transmission of the stimulus to the muscle is not exactly the same from all parts. The duration of latency is inversely proportional to the force of percussion, in normal as well as pathological conditions. The latency is increased in proportion to the number of taps; so that the excitability of the percussed part is diminished by the duration of the experiment. The latency does not differ much in pathological conditions. Rosenheim also found that while the opening and closing shock of the electro-magnetic current was unable to produce the knee-jerk, this could be elicited by the application of the magneto-electric current to the patellar tendon, and also by reversing the direction of a powerful continuous current applied to the same (voltaic alternatives). The phenomena observed by him have led him to the conclusion that no data exist which speak against the knee-jerk being due to reflex action.

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[1] Reichert's and Du Bois-Reymond's "Archiv." Berlin, 1875.

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[1] "The Diagnosis of Diseases of the Spinal Cord," p. 25. Third edition. London, 1884.

[2] "Archiv für Psychiatrie," vol. xv., p. 180. 1884.

It appears to me that an examination of the knee-jerk in cases of fracture of the patella might contribute much to the solution of this question. I lately examined two cases of this kind where partial union had taken place, and which were exhibited by Mr. Christopher Heath and Mr. Morris at a meeting of the Clinical Society of London, and found the knee-jerk equally strong in the limb where the patella had been fractured as in the healthy one. It would, however, be chiefly in fresh cases of fracture of the patella, where there is a considerable interval between the broken fragments, that such an examination might lead to useful results. It stands to reason that percussion of the ligamentum patellæ could not have the same effect in eliciting the knee-jerk in a case of fracture, if the phenomenon was owing to direct stimulation of the muscular substance, as the tension of the quadriceps and the vibratory power of the tendon must be considerably altered under such circumstances; while if the knee-jerk were due to reflex action, little or no change would probably be observed. Hospital surgeons, who have the opportunity of seeing such cases immediately after the accident has occurred, should utilise those occasions by observing the behaviour of the knee-jerk where the fragments of the patella are widely apart, and thus contribute to the settlement of an interesting physiological question. The observations made by myself, in the two cases just alluded to, incline me even more than before to lean to the reflex theory of the phenomenon.

The reflex centre for the knee-jerk is situated in the lumbar portion of the spinal cord, in the region corresponding to the second, third, and fourth lumbar nerves; and the "reflex arc" comprises, on the one hand, the afferent nerves from the patellar tendon, the posterior nerve-roots, and the posterior grey horns; and, on the other hand, the anterior grey horns, the anterior nerve-roots, and the efferent nerves proceeding to the quadriceps muscle. Any interruption of the integrity of this reflex-arc may cause loss of the knee-jerk; and before arriving, simply from the absence of this phenomenon, at a diagnosis of tabes, we must exclude infantile paralysis, pseudo-hypertrophic paralysis, and muscular atrophy owing to neuritis, such as diphtheritic paralysis, etc. For this it is necessary to call in the aid of electricity and mechanical stimulation of the muscular fibres. If there is normal response to faradisation, and if the quadriceps femoris responds by contraction to a tap with the percussion hammer directly applied to its substance, the diseases just mentioned may be excluded. It is a singular fact that in tabes with loss of knee-jerk the mechanical irritability of the quadriceps, more especially of that portion of it which is called the vastus internus, is often increased (Erb, Buzzard), which adds to the value of the symptom. This increase of muscular excitability is probably owing to irritation of the posterior root-fibres, while the loss of the knee-jerk must be owing to destruction of some essential portions of the same. In order therefore to be pathognomonic, the loss of the knee-jerk must be combined with good voluntary power, a proper faradic response, and idio-muscular contraction of the vastus internus.

The physiological and diagnostic importance of the knee-jerk, and the modifications which it undergoes in certain cerebral and spinal diseases, has been almost universally acknowledged immediately after Westphal had brought his researches on this subject before the profession; and it is not too much to say that we owe to this distinguished physician and teacher a debt of gratitude for the new light which the study of these phenomena has thrown upon many obscure diseases of the nervous system. The assistance which we derive day by day in our diagnosis of obscure cases of nervous affections, by utilising these phenomena, is incalculable; and information thus obtained is often of the greatest value. It seems therefore singular that even now this symptom is very little thought of in France. Vulpian, in his otherwise very able lectures on tabes, makes a sneering remark about the discovery "which Westphal thinks he has made;" while Fournier[1] laments the uncertainty of diagnosis in the earliest stages of tabes, when such a symptom is within his reach! The latter author says:—"Il est des malades qui, pendant plusieurs années—trois, cinq, six, même huit et dix années—ne présentent *rien autre* comme phénomènes morbides que des accès plus ou moins espacés des *douleurs fulgurantes*; . . ." and further on (p. 55):—alors même que les malades, s'inquiétant de ces douleurs, viendraient à nous dès leur apparition première, il y aurait encore toutes chances pour qu'elles restassent méconnues quant à leur nature réelle, quant à leur valeur séméïologique . . . Mal décrites sans doute par le patient, vagues de siège, indécises comme caractère, ces douleurs ont été prises pour ce qu'elles n'étaient pas, et rapportées tantôt à de simples névralgies, tantôt à des rhumatismes, tantôt à ceci ou cela, mais *jamais au tabes*."

Loss of the knee-jerk is found in all cases of uncomplicated tabes where we have to do with sclerosis of the posterior columns in the lumbar portion of the cord; and this symptom is of the greatest diagnostic value, more especially in those by no means unfrequent cases where the other symptoms of spinal disease are somewhat indistinct, and it may be overshadowed by signs which are apt to be referred to stomach and liver derangement, or are believed to be owing to gout, hypochondriasis, and other diseases.

There is reason to believe that loss of the knee-jerk is in some cases the first, and in the majority of cases one of the first symptoms of tabes. It was found to have disappeared at a very early stage of the disease in the epidemic of ergotism which has been described by Tuczek, and was in one case the first and only symptom of posterior sclerosis. There is some reason to believe that loss of the knee-jerk may occasionally be preceded by paralysis of the rectus externus or of some other muscle of the eye; but definite data about this are as yet wanting.

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[1] "De l'ataxie locomotrice d'origine syphilitique," p. 50. Paris, 1882.

The following case is of interest in connexion with this point, although the precise date of the loss of the knee-jerk could not be ascertained :—

Case 44.—A country doctor, aged 72, married, who had had thirteen children and forty-nine grandchildren, and had been very successful in practice, consulted me in February, 1882. He had always enjoyed excellent health, and had only quite recently retired from the active duties of the profession. He complained that for some time past he had, on awakening in the morning, been greatly troubled by tingling and numbness in both hands, and more particularly the right. The numbness could be relieved by rubbing, and did not return again during the day, but was always present the first thing in the morning. On examining the patient I found complete absence of the knee-jerk in both sides, with direct response of the vastus internus. He denied having ever had syphilis, but informed me, in answer to my inquiry, that he had had double vision eight years ago, for which he had been treated by Sir William Bowman. This disappeared in about a month, and had never returned. There was no history of any other symptoms of tabes, nor could the closest examination reveal the presence of any of them. The diagnosis might therefore in this case be considered somewhat doubtful; yet the coincidence of double vision, loss of knee-jerk, and numbness is, to say the least, peculiar, and may at some future time be followed by other symptoms of that disease. The patient's age might seem to speak somewhat against the supposition of tabes, but we have seen in the preceding chapter (p. 121) that an advanced age is by no means incompatible with the evolution of the disease.

I have never found the knee-jerk absent in healthy persons, excepting in the two extremes of life. It is difficult or impossible to elicit it in some children before they have learnt to walk, and also in decrepit old persons where there appears to be no particular form of spinal disease (Möbius). The subject of Case 44, however, could not, although of an advanced age, be considered in the least decrepit, as his physical health and his mental energy appeared to be very good.

In the following case the diagnosis of *gout* had been made by several experienced practitioners; and it was chiefly the absence of the knee-jerk which enabled me to recognise the existence of tabes.

Case 45.—A stockbroker, aged 59, married, but childless, consulted me in October, 1881. He complained of "being eaten up with the gout," and having the most fearful gouty pains in his head, chest, back, loins, thigh, stomach, and, worst of all, in the bladder. He had had syphilis fifteen years ago, but had never shown any symptoms of it during the last twelve years. He had lately been treated with citrate of potash, lithia, and colchicum, but without relief. The peculiar description which he gave of the pains to which he was subject made me suspect tabes; and, on examining his legs, I found the knee-jerk absent, which threw at once a flood of light on the nature of his malady. I then examined him for other symptoms of tabes, and found that he staggered on standing with his eyes closed; that he had Argyll-Robertson's symptom of reflectory rigidity of the pupil, could only walk with assistance, had lost the sexual power some years ago, and suffered frequently from incontinence of the bladder and the bowels; he had regular *vesical crises* from time to time, when he would be seized with severe pain in the bladder, chiefly at night, and constant desire to pass his water. On some occasions he had been called out of bed more than sixty times in the course of a single night, and would each time pass only a few drops of water in great agony. He had similar *rectal crises*, and would soil himself five or six times in the course of a night. The upper extremities were not affected, with the exception of an area of numbness in the sphere of the right ulnar nerve.

Case 46.—In July, 1883, Dr. Philpot, of Croydon, asked me to see a gentleman with him, who wished to have an entirely unbiassed opinion on his case. Dr. Philpot therefore told me nothing of the diagnoses which had previously been made, until after I had finished my examination and given my opinion. The patient was fifty-six years of age, married, and father of two children, and had been actively engaged in business. His chief complaint was of depression of spirits, indigestion, and tightness round the chest, which he had had for about two years. A feeling of constriction of the chest being a prominent symptom of tabes, I examined the patellar tendon reflexes, and finding them completely absent, had no difficulty in eliciting the history and presence of numerous other symptoms of that complaint. It appeared that the patient had had syphilis ten years ago. Eight years afterwards, he began to complain of numbness in the third and fourth fingers of both hands; of dull pain in the back and the legs; of incontinence of urine, constipation of the bowels, and loss of sexual desire and power. In addition to these symptoms, there was now reflectory rigidity of the pupils, Romberg's symptom, difficulty in going downstairs, a sensation as if there were a pad of india-rubber under his soles, and slight ataxy in walking.

In the following case the patient complained chiefly of sleeplessness; and the symptoms of tabes were so masked that it would have been impossible to arrive at a correct diagnosis without Westphal's symptom :—

Case 47.—An officer, aged 37, single, consulted me in December, 1881. He had served fourteen years in India, and suffered there during the whole of that time, more or less, from "congestion of the liver" and diarrhœa. He contracted syphilis in 1872. Four months after the appearance of the primary sore, a specific eruption appeared on the legs, but soon faded away again. Soon afterwards he had to complain of soreness on the edges of the tongue and sore-throat; and his tongue has remained permanently sore ever since. For this he was treated by iodide of potassium only; he never had any mercury given him. Soon afterwards he began to suffer from severe pains about the loins, which he attributed to having strained the muscles of his back while playing rackets. He played this game daily while in India, and also indulged freely in promiscuous sexual intercourse. In 1878 he was obliged to give up rackets, and had since then had constant pain in the loins, and that in any position, whether sitting or lying, standing, walking, or riding. Sixteen months ago insomnia came on, and proved most obstinate. At the same time he began to feel a distaste for sexual indulgence, society, and his duties. He also had a feeling of "fierce compression" across the chest. He returned to England, and remained here for six weeks, after which he went back to India. Soon after his arrival there, a feeling of intense spinal irritation came on, which extended up and down the entire length of the spine, and was accompanied by numbness and "pins and needles" in the hands and feet. The irritation in the back led to great nervousness; the patient also complained of overpowering lassitude and weakness in the legs, and felt it impossible to continue his duties. He once more returned to England in April, 1881, and was put under mercurial treatment, which however did him no good. In August he went to

Gastein, took twenty-seven baths, and was galvanised, also without any effect. When I saw him, his chief complaint was of insomnia and great hyperæsthesia of the back. The latter symptom induced me to examine the patellar reflexes, which I found to be absent in both sides. I now inquired for other symptoms of tabes, and found that he had numbness in all four extremities ; considerable debility in walking, although no ataxic gait; the muscles of the legs were thin and flabby ; and the sexual power and desire were greatly diminished.

While, therefore, the loss of the knee-jerk habitually indicates tabes spinalis, even where other symptoms seem to point in a different direction, its *presence* or *exaggeration* will show us that although many symptoms of tabes may be present, yet the case is not one of locomotor ataxy or sclerosis of the posterior columns of the cord, as generally understood. In such cases which are far from uncommon, we have rather to do with a combined system-disease of the lateral and posterior columns, or with disseminated insular sclerosis, than with true tabes. The exaggeration of the knee-jerk is then to be explained by the sclerotic irritation of the lateral columns, which is too powerful in its effects to be neutralised by the disease of the posterior columns. Lightning pains are mostly absent, and the difficulty in walking is rather owing to paresis than to ataxy, or to a combination of these two conditions.

Case 48.—A miller, aged thirty-one, married, and father of two children, was admitted into the hospital under my care, in February, 1883. He denied any syphilitic infection, and attributed his illness to severe chills which he had habitually experienced in his business. Twelve years ago he had broken his right leg, and there was now a large exostosis on the tibia which, but for this history, might have been looked upon as of a syphilitic character. His present illness commenced three years ago, when he lost his taste and had double vision. At the same time he found that he could not walk so well as before, more especially in the dark. After a time the sense of taste gradually came back, and he saw things single again; and two months after having been first taken ill, the power in the legs improved so much that he thought he walked as well as ever. Twelve months ago, however, he began to get worse again, and the disease has since then gradually gained upon him. On examination the brain and cranial nerves appeared to be healthy. The patient complained of a sensation as if he had a tight rope round his chest, of difficulty in retaining the urine, obstinate constipation, and complete loss of sexual power. He felt numbness in the hands, and some degree of ataxy in the upper extremities, more especially when he attempted to write. His gait appeared to be a mixture of ataxy and paresis, as there was decided loss of muscular force, together with jerkiness; the patient had to manœuvre with his arms while walking, as otherwise he felt too unsteady to go on; and on shutting his eyes he staggered to such an extent that he would have fallen unless supported. He also complained of numbness in the soles of the feet; but he had never had lightning pains, or indeed any other kind of pain. On testing the deep reflexes, I found them all very much exaggerated; and I therefore concluded that the case was not one of tabes spinalis, but one of an unusual form of insular sclerosis of the spinal cord, which had affected several strands of fibres in that organ, but had spared those which are more particularly concerned with the sensation of pain and the conduction of reflex action in the posterior columns.

The absence of lightning-pains is, however, not invariable in such cases, as shown by the following instance:—

Case 49.—An Irish farmer, aged fifty-one, married, and father of two children, consulted me in October, 1882. He complained of incontinence of urine, loss of sexual power, obstinate constipation of the bowels, inability to walk in the dark, numbness, and *occasional attacks of lightning-*

pains in the legs, the muscles of which were exceedingly flabby and wasted. He had had syphilis rather badly ten years ago. He was seen to stagger when standing with his eyes closed, and his gait was a mixture of ataxy and paresis. The patellar reflexes were greatly exaggerated, and I therefore concluded the case to be one of combined system disease of the lateral and posterior columns.

Case 50.—Another case in which there was a marked combination of loss of power and ataxy, was that of an accountant, aged thirty-two, single, who was sent to me in October, 1879, by Dr. Haussmann, of Wildbad. This patient had "knocked about town" a good deal about ten years ago, and had then had a chancre which was followed by a large crop of "secondaries," chiefly on the skin and in the throat. Two years afterwards he had repeated attacks of hemiplegia and aphasia, and was inefficiently treated in Java, the Cape, St. Helena, and various places. He got better and worse, off and on, but about three years ago began to suffer from more definite symptoms of spinal disease. He had now the greatest difficulty in getting up from a chair, in standing and walking. There was great loss of muscular power, more particularly in the right leg. The bladder was so irritable, that he felt constant desire to pass his water, and he wetted the bed in his sleep. The bowels were obstinately constipated; the sexual power almost entirely lost. The knee-jerk was greatly exaggerated in both sides; and it was therefore evident that the case was one of combined system-disease of the cord. In this case, likewise, no lightning pains had occurred at any time.

Does the knee-jerk, when once lost, ever come back? There is very little positive experience about this, but a great deal of negative evidence. In general, I may say that *it never returns* in cases of ordinary tabes; but I have seen it return, under the influence of treatment, in cases of combined cerebral and spinal disease, which presented themselves under the clinical aspects of general paralysis of the insane in the first period of the complaint, more especially where this was owing to syphilis. In a case of this kind, in which I was consulted in April, 1884, and where the absence of the knee-jerk, together with grave cerebral symptoms, appeared to afford a gloomy prognosis, the patellar reflex returned, under the influence of an energetic specific treatment, within a fortnight, together with great improvement in other respects. I have at present (May, 1884) a patient suffering from the effects of injury to the spine under my care, at the hospital, in whom the accident had caused paresis of the lower extremities. The knee-jerk was at first exaggerated, but, as time went on, became more and more sluggish, and eventually vanished altogether. The patient, who had at first been treated with iodide of potassium, was now put on full doses of ergot; and, after he had taken this for six weeks, the reflex could again be elicited. At the same time there had been proportionate improvement in the motor power of the lower extremities all the time that the patient was taking the ergot.

With regard to this point Westphal has drawn attention to a source of error, which consists of confounding certain skin-reflexes with the true knee-jerk. Contraction of the quadriceps femoris may occasionally be caused by pinching a fold of the skin in the neighbourhood of the patellar tendon, or percussing it sideways, without touching the patellar tendon itself. In some persons all the muscles of the thigh are seen to contract if the skin above the internal malleolus be strongly pinched. There is, therefore, a "pseudo-knee-jerk;" and whenever it is reported that the knee-jerk has returned, it must be plainly stated that the observer has not been deceived by cutaneous reflexes.

Eulenburg and Strümpell have seen the patellar reflex return after strychnia injections. Dowse[1] believes that he has "restored the lost knee-jerk *instanter* by passing a continuous current freely though the spinal cord." This is a startling assertion, but hardly less so than another statement by the same author, "that the knee-jerk and all other reflexes are not uncommonly exaggerated in the earlier stage of tabes, and that this may be accounted for by the possible probability (*sic*) that the changes in the posterior root-zones are extremely peripheral, and invade more or less the adjacent matter of the lateral columns." Tabes is here evidently confounded with insular sclerosis, or some other form of combined system-disease of the cord; while the "instantaneous recovery of the knee-jerk by the passage of an electric current through the spine" would presuppose an immediate cure by that means of a definite anatomical lesion in the lumbar portion of the cord, which is, unfortunately for our patients, too stubborn in character to yield, so readily as has been assumed, to a single application of electricity.

Loss of the knee-jerk on *one* side appears to be extremely rare. Even where the other clinical symptoms have been entirely limited to one side of the body, thus constituting a kind of *hemi-ataxy*, I have invariably found the loss of the knee-jerk to be bilateral, even at an early period of the malady. A few other observers have, however, apparently seen the phenomenon absent on one side, and present on the other.

2. *Lightning-Pains.*—This term is well suited for describing the exceedingly violent and sudden nature of the pain which is so commonly experienced in tabes. Synonyms are "lancinating" and "fulgurating" pains. Some patients compare these sensations to electric shocks; others describe them as violent blows with a heavy hammer, or kicks, or nails driven into the flesh, or

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[1] "Medical Times and Gazette," October 1, 1881.

as if a red-hot corkscrew were turned round in the limbs, or like a sudden contact with a red-hot iron, or as if they were bitten by dogs, gnawed by rats, or as if the flesh were forcibly torn from their bones, or as if their bones were crushed in a press, or their insides burnt with fire, or as if they were actually being impaled, or as if they were struck by a dagger or stabbed with a knife, or as if thousands of fish-hooks had got hold of the calves of the legs, and were dragging them up. Pains of this kind are apt to come on suddenly, chiefly at night, and in paroxysms, so that a shock is experienced every few seconds or every few minutes; and they vanish after a variable time as quickly, and often without any apparent cause. In their greatest degree of violence they constitute the most frightful torture which can be imagined, and which renders life utterly wretched and intolerable. The patients actually writhe with pain, scream, call for mercy, twist themselves into various positions, squeeze the painful spots as hard as they can, and, when the attack is over, seem to be half-dead from exhaustion. As such paroxysms occur chiefly at night, they prevent the patient from sleeping, and thus aggravate the general disturbance of the system which is the consequence.

Charcot distinguishes lightning pains, which run like lightning shocks through the course of a nerve or a limb; and *terebrating* or boring pains, which are of the dagger-and-cork-screw character, and mostly occur near a joint. This distinction, however, is imperceptible in a number of cases, as both forms may occur together.

Another form of pain is less violent and more constant. This is generally fixed in a certain locality. For instance, there is a dull back-ache, a pain between the shoulders or in the spine, and spreading from there to the sides of the body; or both legs suffer in the same manner, so that the patient is believed to have bilateral sciatica. This aching may also come on in crises, and continue for a few days or weeks. Occasionally the pain is not really constant, but consists of a series of acute paroxysms following each other rapidly. The more paroxysmal and the more constant pain may alternate in the same patient.

A third kind of pain, which is, according to Fournier, chiefly felt by syphilitic subjects, is of a much slighter character, and comes at longer intervals. This resembles a slight prick with a pin, apparently hardly going beyond the epidermis, or a slight pinching of the flesh. It is so insignificant that patients take little notice of it, but remember it afterwards, when they may be suffering from more severe paroxysms. There are often long intervals between these slight attacks, which come on for a few hours only, once in two or three or even six months. Unless a specific treatment be instituted, however, the pain is liable to become much worse as time goes on, and to occur at much shorter intervals.

The principal seat of all these different kinds of pain is in the lower extremities, where they are almost invariably felt in the commencement of the disease, and where they may continue to rage with unabated fury from time to time for years. They may shoot all the way down from the hips to the toes, or follow a certain nervous distribution, as in neuralgia, or affect a limited area, such as the ankle, the knee, thigh, or hip. If they are localised in a small space, hyperæsthesia is apt to supervene in the same, so that the least touch is exceedingly painful, while strong pressure is apt to relieve the pain. Occasionally an erythematous rash or circumscribed urticaria breaks out in such a spot after the pain has been going on for some time ; and Charcot has seen ecthyma in the sphere of the crural and internal saphenous nerve, leaving scars which have been visible for years. Muscular twitches in the neighbourhood testify to the increased reflex excitability which is gradually established. Occasionally a shock through the legs is so sudden that the patient, if walking or standing at the time, drops down as if he were shot, and is believed to have a fit. Sometimes the pain affects, even in the beginning, other parts than the lower limbs, such as the back, the upper extremities, the body, and the head. When affecting the chest, it often resembles intercostal neuralgia, or the pain of herpes zoster. In a considerable number of cases the pain appears to follow very closely the anatomical changes going on in the cord ; and as these are apt to spread from below upwards, so the pain, after having been for some time confined to the lower extremities, gradually invades the abdomen, chest, the upper extremities, and the head. It is sometimes confined to one limb or one side of the body, and sometimes to the upper extremities. In such cases the other symptoms of tabes which follow in the further course of the disease are apt to show a similar distribution, showing that they follow pretty closely the anatomical process as it spreads further in the substance of the cord. In the upper extremities the area of the ulnar nerve is affected by preference, the shocks running from the olecranon down into the third and fourth finger.

Pains in the face and head, to the occurrence of which in tabes Pierret[1] was the first to draw attention, present the same character as the pains in the limbs, being either paroxysmal, in the form of lightning pains, or more continuous. The latter are by far the more frequent, and affect the top of the head, the temples, the bridge of the nose, the auditory meatus, the jaw, lips, and teeth, or the occipital region. Some patients have a sensation as if the eye were suddenly torn from its socket, or feel a sudden stab or shock right through the head or in the orbit. This may be accompanied with lacrymation, photophobia, and certain vaso-motor symptoms, such as congestion of the conjunctiva, dilatation of the pupil, and elevation of temperature ; others see flames and flashes of light, showing that the optic nerve participates in the irritation. Sometimes the pain is confined to one side of the head, and resembles megrim. It is rebellious to treatment, often lasts a long time, and then suddenly disappears without any apparent cause. It is therefore important to remember, in cases of what is called facial neuralgia, that this may be an early symptom of tabes.

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[1] "Essai sur les Symptômes céphaliques du Tabes dorsalis," p. 21. Paris, 1876.

We have seen (p. 28) that the nucleus of the fifth nerve has been found wasted in tabes by Hayem and Pierret, but that it is as yet uncertain whether this nerve suffers in its peripheral course. Pierret explains the pain which is, in tabes, experienced in the sphere of the fifth nerve, by sclerosis of the descending root of the same, which may be traced down in the cord as far as the level of the third or fourth cervical nerve ; while the pain at the back of the head, in the sphere of the occipital nerve, would be accounted for by this nerve being the internal branch of the posterior division of the second cervical nerve. Thus, the lesion causing these pains would be central, bilateral, and symmetrical, and this would be in accordance with the fact that the facial neuralgia of tabes is not generally so confined to a single branch of the trifacial as it is in idiopathic neuralgia, but may play about in the entire area supplied by the fifth pair.

In general, pains of extreme violence are temporary, but apt to recur at short intervals. In this respect there are the greatest possible varieties in individual cases. Occasionally the intervals are very long, amounting to six months and more; and there is generally an exciting cause to be discovered for a " bout of pain." In this country it is chiefly the easterly winds of March and April, and a sudden change to wet weather at any time of the year, or thunderstorms, which are dreaded by tabid patients. Other exciting causes are sudden emotions, more especially annoyance, over-exertion, sexual indulgence, and nocturnal seminal emissions. In exceptional cases pain of any sort may be absent throughout the first stage of the disease; or it may be so slight that the patient forgets all about it, or simply says that he may have had a little rheumatism.

Topinard has found these pains altogether absent in twenty-two cases out of a hundred and four; and Erb in eight cases out of sixty. According to my experience they are almost invariably present in really well-marked cases of tabes, while they are absent in those patients who, although presenting many symptoms of sclerosis of the posterior columns, appear to be subject to combined system-diseases of the cord, or insular sclerosis. I have already drawn attention to the circumstance that, where the knee-jerk is exaggerated instead of being lost, lightning pains are habitually absent (p. 142).

On the other hand, we find that they are not absolutely limited to tabes, but that they may occur whenever the posterior columns, and more particularly Burdach's strands, are affected. Thus they have been observed in myelitis and meningo-myelitis, where this was either spontaneous or consecutive to Pott's disease of the vertebræ; in some forms of insular sclerosis, of general paralysis of the insane, of chronic alcoholism, and in alcoholic paraplegia. Again, in certain forms of sciatica and tic-douloureux, the pain is so similar to lightning pains, that it would be impossible, from this symptom alone, to make a distinction. In such cases the seat of the neuralgia is probably more central than peripheral, and located in the posterior nerve-roots.

Charcot was the first to express the opinion that the lightning-pains in tabes are owing to sudden irritation of the posterior nerve-roots, in the root-zones of the posterior columns; and this explanation is so plausible that it has been generally accepted. We could, with this theory, easily account for the varying degrees of intensity of the pain, by assuming a corresponding degree of irritation in Burdach's columns ; while the differences in the seat of the pain would be explained by the different areas of root-fibres in the cord which might happen to be subject to irritation. That the pain should not be felt so much in any portion of the back as in the peripheral expansions of the spinal nerves is, as Vulpian has appositely remarked, in accordance with the general law, which ordains that impressions produced in the cord, or at any portion of the nerve, are by the brain referred to the periphery. Pain, although produced in the centre, is felt in the skin, and sometimes in such a limited area of the skin that the patient is loth to believe that it really arises from irritation in the spinal cord, when he feels no pain whatever in the spine. This, however, is likewise the case in certain forms of neuralgia, such as tic, intercostal neuralgia, and sciatica, where the pain is produced in the nervous centres, or the nerve-roots, or the spinal canal, or the skull, while it is perceived peripherally. This will explain why neurotomy and neurectomy of the apparently affected nerve are so often unsuccessful.

The theory of the central production of lightning-pains is apparently contradicted by the fact that these pains may be relieved by certain local applications to those parts of the skin in which they are experienced. Such applications may be of the most varied character ; the chief amongst them being counter-irritants, such as blisters, sinapisms, hot fomentations, and chloroform, subcutaneous injections of morphia and atropia, and, what seems more extraordinary still, injections of plain water into the cellular tissue. This contradiction, however, is only apparent, as will be seen from the following considerations:—

It is a well-known fact that the pains of tabes are apt to be made worse by a slight touch of the parts in which they are experienced, while strong pressure, on the contrary, gives relief. The tabid patient, when subject to a paroxysm of lightning-pains, is therefore generally seen to squeeze the parts affected as hard as he is able. In order to understand this, we must remember that waves of nervous influence are constantly transmitted from the peripheral expansions of the sentient nerves to the nervous centres; and these play, unquestionably, a considerable part in the causation of the pains of tabes. We know that changes in the weather, a thunderstorm, or the simple influence of cold, will give rise to a paroxysm of lightning-pains. In a healthy person the influence of such waves of nervous force passes, in general, unperceived; but when nervous elements are traversed which are in a state of morbid irritation, pain is caused. Nothing can be more intimate than the relation which exists between the suffering posterior nerve-roots, on the one hand, and the peripheral sentient nerves in the skin, the subcutaneous cellular tissue, the muscles, and even the periosteum of those parts where the pain is experienced, on the other hand. An injection of morphia and atropia has no doubt a general influence in subduing undue excitability in the posterior columns and nerve-roots; but its power of relieving the pains of ataxy is also to some extent owing to a kind of local anæsthesia which is caused in the peripheral fibres near which the injection is made. The fibres which are in direct contact with the liquid injected, are being temporarily paralysed by the narcotic; and complete or incomplete anæsthesia of the skin, in a circumference of an inch or two in diameter, may be found there, and may last for several minutes. In consequence of this, the waves of nervous irritation, which were transmitted by those fibres to the suffering centres, are for the time being diminished or suppressed. Hence it results that the paroxysm ceases in the centre, and the pain disappears. The pain may not even return when the local anæsthesia in the area of injection has disappeared, partly because the irritation of the roots has ceased, and partly because the general effect of the morphia and atropia, which now arrive with the blood at the suffering parts, is superadded to the local action.

Ranvier has shown that when a nerve of an animal is laid bare and subjected to the action of plain water for some time, say a few minutes, it loses its physiological properties, probably from swelling of the axis-cylinders of the nerve-fibre through the imbibition of fluid. It is no doubt in a similar way that injections of plain water into the cellular tissue of a region affected with pain are effective in relieving the latter. The effect is therefore not simply owing to the imagination of the patient, as has been generally supposed ; but the afferent nerve-fibres, bathed in water, lose for a time their excitability and conducting power, and therefore cease to transmit waves of irritation to the centres.

The influence of chloroform and other counter-irritants in relieving the lightning-pains has to be differently explained. It is a suggestive fact that such substances produce their effect chiefly when they cause a considerable degree of local irritation and rubefaction, and much less so when the skin—as is the case in the later stages of tabes—has become anæsthetic, and when the local effect which is produced is less powerful or altogether absent. It has been stated that counter-irritation of the skin produces a contraction of the blood-vessels in those portions of the posterior roots and columns which are in special relation with the peripheral nerves acted upon, and that the anæmia thus caused in the suffering parts, tends to relieve the irritation. This, however, is pure speculation. Brown-Séquard believes that he has produced contraction of the arterioles of the spinal pia mater by irritation of the supra-renal capsules; but subsequent observers have been unable to corroborate his results; and even supposing that such a contraction took place, this would not prove that the irritation of the posterior roots and columns could thereby be diminished or arrested. A far more plausible explanation is that given by Vulpian, viz., that the pain is relieved owing to a powerful impression being produced on the peripheral cutaneous nerves, which the latter transmit to the posterior roots and columns, thereby profoundly modifying their condition. Whether this modification of the molecular state of the central parts is more or less permanent or temporary must depend upon individual circumstances. It will, however, be seen from the foregoing considerations that when we find local applications useful in cases of neuralgia, in the anæmic, the hysterical, the rheumatic, the syphilitic, and the alcoholised, this result does by no means prove that the seat of the pain was local, but, on the contrary, that the seat of the pain is likely to be central rather than peripheral.

Lightning-pains are also apt to occur in diverse viscera, constituting gastric, laryngeal, vesical, rectal, and other "crises"; but as, in accordance with the localisation of these pains, a great variety of other symptoms are observed, I consider it preferable to discuss these symptoms separately, further on.

3. *Reflectory Rigidity of the Pupil* (*Argyll-Robertson's Symptom*).—The condition of the iris varies considerably in the initial stage of tabes. Quite in the beginning the pupils are apt to be rather large than small. Mydriasis in one eye I have known to be one of the first symptoms of the disease, but the most peculiar and characteristic change which occurs in the pupils in this malady is that which was first described by Argyll-Robertson,[1] of Edinburgh, and, therefore, bears his name. It consists of bilateral myosis, or contraction of the pupils, with loss of reflex action to the influence of light, but with preserved voluntary contraction during accommodation. The symptom is, therefore, seen to be closely analogous to the loss of the knee-jerk, where reflex action is lost, while voluntary power remains, and which we have seen to be a sign of the first importance in tabes.

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[1] "Edinburgh Medical Journal," p. 696, Feb. 1869.

Argyll-Robertson has placed on record the case of a carver, aged fifty-nine, who was then evidently in the second stage of tabes, and in whom he noticed that both pupils were contracted to little more than pin-points, and did not contract under the influence of light, but did so when the patient was told to accommodate the eyes for a near object. The pupils dilated only slightly under the influence of a four-grain solution of atropine ; and although this was re-applied the next day, it failed to produce any further change ; its action, moreover, was transient, even the limited dilatation soon disappearing. Calabar bean, on the other hand, produced further contraction of the pupil, reducing its diameter to rather less than one-fourth of a line, which is more than is produced in other cases by the same agent. The right eye was quite colour-blind ; but with the left the patient could distinguish blue, and generally the other colours when of a bright tint, although even then he made occasional mistakes, having a tendency to call all colours of which he was uncertain yellow or gilt. This symptom had been a source of annoyance to the man, as it prevented him from distinguishing different woods, such as birch and mahogany. Neither eye was abnormally sensitive to light, and vision was best in bright daylight. His field of vision was, however, markedly contracted in both eyes.

Argyll-Robertson attributed this condition to disease of the cord affecting the cilio-spinal region, and thought it owing not to spasm of the sphincter, but to paralysis of the dilator muscle or the radiating fibres of the iris. He was not the first to observe myosis in cases of tabes ; for Romberg had previously noticed that in such patients the pupils are often contracted to the size of a pin's-head ; while Trousseau had seen the same, and also found this contraction to resist the influence of belladonna, and that during paroxysms of pain the contraction was replaced by more or less dilatation of the pupil.

The symptom which we are now discussing appears to be very frequent. Vincent[1] found morbid changes in the pupils in forty-seven out of fifty-one tabid patients (or ninety-two per cent.), the action of the iris having been normal in only four cases. Of the forty-seven there were forty in which the pupils had lost the light reflex, but acted during accommodation, while in seven they were completely immovable. Of the forty cases where the light-reflex was lost, there were twenty-three with myosis, six with mydriasis, and eleven with normal size of the pupils. The percentage for Argyll-Robertson's symptom was, therefore, 46·6. With regard to the different stages of the malady, he found that in the initial period the pupils are frequently enlarged, and do not respond to the influence of light, but to accommodation ; that in the second period there is generally myosis and no response to light, but that the pupils contract for near objects and dilate for far objects ; while in the third or terminal period the pupils are generally dilated or of normal size, and completely motionless. In nine cases of other diseases of the cord and the brain he found good response to light eight times ; while in twenty-one cases of general paralysis of the insane he found loss of the light-reflex, and preserved contraction during accommodation nineteen times; in seventeen cases, however, the pupils were unequal, while there was myosis in eight, and slight mydriasis in three. While, therefore, tabes and general paralysis have the loss of the light-reflex in common, they differ widely from one another by the pupils being generally of equal size in tabes and unequal in general paralysis.

Erb[2] has seen spinal myosis in sixteen cases out of thirty (54 per cent.), and I have seen it thirty-two times in fifty consecutive cases (60·4 per cent.).

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[1] "Des Phénomènes oculo-pupillaires dans l'Ataxie locomotrice." Thèse de Paris, 1877.

[2] "Deutsches Archiv für klinische Medicin," vol. xxiv., p. 31. Leipzig, 1879.

Where the pupil is much contracted, there are often other signs of paralysis of the vasomotors: such as a red cheek, an injected conjunctiva, and increased temperature of the face. During paroxysms of pain, the contracted pupil, however, becomes dilated, and the symptoms of vasomotor paralysis disappear. The ordinarily unexcitable pupil may be excited reflexly if one eye is closed and its lid lubricated with the tip of the forefinger and pressed; then the pupil of the opposite eye is seen to dilate considerably. The same occurs when the skin of the temples, or mastoid processes is faradised with the brush.

The exact mode of production of Argyll-Robertson's symptom is not yet satisfactorily explained. It seems quite clear that the pupils are contracted from paralysis of the radiating fibres of the iris, or dilators of the pupils, while the circular fibres of the iris, or sphincters of the pupils, remain intact, and therefore show predominance of action. If there were actual spasm of the circular fibres, the maximum of contraction of the pupils would be reached, which is not the case, seeing that they may be made to contract still further, both during efforts at accommodation and by the influence of Calabar bean. Now the circular fibres are innervated by the third nerve, while the radiating fibres are supplied by the sympathetic. Paralytic myosis may therefore arise from disease of the sympathetic root of the lenticular ganglion, or from tumours pressing on the cervical sympathetic nerve, or from disease of the cilio-spinal region of the cord. It appears, *primâ facie*, reasonable to assume that bi-lateral myosis in tabes is owing to the latter affection; and Remak sen. had already, in 1864, drawn attention to the occurrence of myosis in cervical tabes. Disease of the spinal centre for the dilatation of the pupil, or of the fibres proceeding from it, is therefore likely to be at the root of paralytic myosis.

Hempel,[1] who has given great attention to this question, believes that the myosis is owing to paralysis of the centre for pupillary dilatation in the medulla oblongata, and that the loss of the light-reflex is owing to an interruption of the reflex arc between the optic and the oculomotor nerve, the centre of the latter being normal.

Vincent, on the other hand, considers that the reflectory paths which proceed from the optic nerve descend into the cervical cord, and there undergo a fusion with motor centres, from which again other paths proceed, which stimulate the sphincter of the pupil, to become afterwards connected with the trunk of the third nerve, and thus to arrive at the iris. The loss of the light-reflex would therefore be owing to disease of certain centres and paths which are situated in the cervical portion of the cord.

Recent experiments of Bechterew have shown that the reflectory centre for the nerve-fibres which constrict the pupil is situated in the nucleus of the third nerve; for destruction of this nucleus as well as division of the third nerve, causes maximal dilatation of the corresponding pupil, and complete immobility of the same to direct or indirect stimulation by light. On the other hand, destruction of the optic tract, the corpora geniculata and quadrigemina, destroyed the function of the retina, but had no influence on the mobility of the iris. Each pupil appears to have an independent reflectory arch for itself; the fibres which constrict it, proceeding from the retina to the optic nerve, coursing in the latter, entering behind the chiasma immediately into the central grey matter surrounding the cavity of the third ventricle, proceeding thence to the nuclei of the oculo-motor nerves, and returning in the sheath of the latter to the periphery. Commissural fibres between the nuclei of the third pair connect the two arches, so that the reflex may be carried from one eye to the pupil of the other. When the pupil is dilated in consequence of painful stimulation, this is not owing to the action of sympathetic fibres, but is caused independently, by inhibition of the light-reflex.

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[1] "Archiv für Ophthalmologie," vol. xxii., p. 1. Berlin, 1876.

Reflectory pupillary rigidity would, therefore, according to this be caused by lesions interrupting the path of the light-reflex in its course from the chiasma to the nucleus of the third nerve.

The presence of the three symptoms which I have just described, viz., loss of knee-jerk, lightning-pains, and reflectory rigidity of the pupil, render the diagnosis of tabes perfectly certain, as such a combination does not occur in any other disease. In some cases, however, either one or both of these latter symptoms may be absent; and we have then to look for other evidences of the malady, which I shall now proceed to consider.

4. *Mydriasis and Paralysis of the Ciliary Muscle.*—Paralytic mydriasis constitutes that state of dilated pupil which results from paralysis of the circular fibres of the iris, while the radiating fibres of that membrane remain intact. It is owing to injury or disease involving the third or oculo-motor nerve, either in its nucleus or in its course towards the orbit; and is generally seen in certain diseases of the brain where this nerve becomes implicated, such as meningitis, hydrocephalus, etc.; or it may be a symptom of inflammation or sclerosis of the nerve itself, or of disease in the short root of the ciliary ganglion. Mydriasis is generally combined with paralysis of the ciliary muscle, causing loss of accommodation; and the impairment of vision which results is then chiefly owing to the latter.

Paralytic mydriasis may be one of the first symptoms of tabes, and precede the appearance of lightning-pains and other symptoms by several years. If it comes on suddenly, and without any apparent cause, such as severe exposure to cold, injury to the eye, etc., it is generally of a syphilitic nature; and if combined with loss of the knee-jerk may be looked upon as foreshadowing the outbreak of tabes.

Case 51. In October, 1867, Mr. White Cooper requested me to see a gentleman, aged thirty-four, married, who had had syphilis very badly ten years ago, but had for some time past been free from symptoms of it. Twelve months before I saw him he had been struck by lightning. In January last, however, affection of the iris came on quite suddenly, as he found one morning on attempting to read that "all the letters danced about like Hebrew," and the right pupil was much dilated. The degree of this dilatation varied at first, but since April last had become stationary. Calabar bean and a variety of other remedial measures produced no effect. When I saw him, the pupil had an ovoid shape, and was almost, though not quite, as much dilated as if it had been under the influence of atropine. It was quite immovable, and did not respond to light or to efforts of accommodation. The sight was considerably impaired, which was no doubt more owing to the loss of accommodation than simply to the dilated pupil.

This case occurred before the days of "tendon-reflexes," so that the condition of the knee-jerk was not ascertained. Ten years afterwards, however, the phenomena of locomotor ataxy had become fully developed in this patient.

5. *Other Palsies of Ocular Muscles.*—One of the commonest symptoms of tabes is *temporary double vision,* owing to paresis or paralysis of the rectus externus or rectus internus muscles. Such double vision may last only a few hours and then vanish and occur again a few months afterwards. In other cases it may last for a few weeks or a month, then disappear, apparently under the influence of treatment, and return again six months afterwards, etc. Some patients have six or seven such attacks, and eventually the palsy may become permanent.

a. *Paralysis of the sixth nerve or abducens* is a very common occurrence in tabes, and causes convergent squint. The degrees of this affection are exceedingly variable, as in some cases it is so slight that it can only be discovered by the most careful examination, while in others it is so severe that the eye cannot be moved outward beyond the middle line, and the inward deviation of the eye during rest may be so considerable that the cornea is entirely concealed at the inner angle of the orbit. In most cases which I have seen in connection with tabes, the paralysis of the sixth nerve occurred on the left side.

Double vision then appears when the object is moved into the left half of the visual field, but is absent in the right half ; and the further the object is moved to the left, the greater will be the distance between the double images. In consequence of the confusion caused by the double images, severe giddiness and even vomiting may be caused ; and the efforts which the patient makes in order to correct the diplopia, by increasing the action of the superior rectus and inferior oblique, often lead to considerable pain and exhaustion. Duchenne has seen cases of bilateral affection of the two sixth nerves in which there was no double vision. This affection may also be intermittent, occurring every other day.

b. *Paralysis of the ocular muscles supplied by the third or oculo-motor nerve* is likewise frequent. *Ptosis* of the upper eyelid generally occurs together with paralysis of the superior rectus, and may be complete, when the eye appears entirely closed ; but is more frequently incomplete when the palpebral fissure appears to be more or less narrowed, in exact proportion to the degree of loss of power in the levator palpebral superioris muscle. Ptosis is generally a more permanent symptom than paralysis of the rectus externus, and less likely to disappear without some definite treatment, more especially an early application of the continuous current, being undertaken.

Paralysis of the internal rectus causes divergent squint; the eye is pulled outwards by the unrestrained action of the external rectus, and cannot be moved beyond the median line if the paralysis is complete. In paralysis of the left rectus internus, the false image is to the patient's right, and the distance between the two images increases the further the object is moved towards the right side.

Paralysis of the fourth or trochlearis nerve by itself is very rare in tabes.

c. *External ophthalmoplegia*, or symmetrical immobility of all the external ocular muscles is likewise a rare occurrence. This affection was first described by the founder of modern ophthalmology, A. von Graefe,[1] and afterwards by Jonathan Hutchinson.[2] The first symptom is drooping of both eyelids, so as to give the face a sleepy expression; and this is soon followed by paresis or paralysis of all the ocular muscles, so that the movements of the eyeball are restricted or even wholly lost. The power in the muscles fails in groups, and not singly; and paresis is more frequent than paralysis. There is seldom complete ptosis, although the eyelids droop; and impaired range of the motion of the eyeballs is more common than absolute fixation. In some of these cases it seems that there is no actual paralysis, but that the co-ordination or synergy of the different groups of ocular muscles has been destroyed. A patient who shows symptoms of paralysis of the third nerve on one side when using both eyes, will be able to move the affected eye and eyelid much better when using only that eye, and putting the other for the time being out of action. At a late period, however, the paralysis may be absolute. The third, fourth, and sixth nerves are then all involved, and the eyeball is then found to be completely immovable, and to stand in the middle of the palpebral fissure, in a forward direction, covered by the drooping upper eyelid. There are double images in all directions where the paralysis is unilateral, and where vision is preserved in the paralysed eye. The pupil is moderately enlarged, and the accommodation for near things diminished or lost. Exophthalmus is usually present. There may be diverse combinations, so that the third and the sixth nerve, or the third and the fourth, or the fourth on one side, and the sixth on the other side, are paralysed together.

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[1] "Archiv für Ophthalmologie," vol. xii., pt. 2, p. 265. Berlin, 1866.

[2] "Medico-Chirurgical Transactions," vol. lxii., p. 307. London, 1879.

The mode of onset of these combined palsies is only exceptionally acute, but mostly slow, and their evolution may be accompanied with headache, giddiness, and confusion. In some cases other cranial nerves, such as the olfactory, the optic, the motor portion of the fifth, the portio dura, and the pneumogastric are likewise affected. Von Graefe found that some of these cases occurred suddenly in consequence of exposure to cold, while in others it was probably owing to syphilis; and Hutchinson discovered that some patients suffered from locomotor ataxy. In one of them, where an inspection was obtained, degenerative atrophy was found in the optic, third, fourth, and sixth nerves, and the ganglionic cells had disappeared from the nuclei of all the affected nerves. The spinal cord could not be examined, but the symptoms observed during life —more particularly a sensation of tightness round the abdomen, numbness in the hands and feet, incontinence of urine, obstinate constipation of the bowels, and eventually paraplegia—can leave no doubt whatever on the mind that, had the cord been examined, posterior sclerosis would have been discovered. One of Hutchinson's patients became eventually insane, and another was before death liable to attacks of violent mental excitement, showing that tabes had become complicated with general paralysis of the insane.

The affection has therefore the greatest possible resemblance to labio-glosso-laryngeal paralysis, in which there is also degenerative atrophy of the ganglionic cells of the nerve-nuclei, on the floor of the fourth ventricle ; yet this latter affection does not occur in tabes, and appears generally to have no relation to syphilis.

d. *Internal ophthalmoplegia* is a group of symptoms which was first described by Hutchinson,[1] and includes according to him, *iridoplegia*, or paralysis of the circular and radiating fibres of the iris, and *cycloplegia*, or paralysis of the ciliary muscle. In iridoplegia we have to do with total paralysis of the iris, of its circular or contracting as well of its radiating or dilator fibres, causing the pupil to be perfectly motionless, so that it is quite insensible to the stimulus of light, and can neither be contracted nor dilated. In cycloplegia, on the other hand, we have total loss of the power of accommodation, so that young and middle-aged persons require strong convex glasses for reading. This latter occurs by itself only after diphtheria, but in all other conditions appears to be combined with iridoplegia. Syphilis appears to be the only cause which could be discovered in Hutchinson's cases. This able observer does not mention that any of his patients were likewise suffering from tabes; yet there can be no doubt from his description of some of their symptoms that at least some of them were affected with that disease. Thus it is mentioned that one patient had suffered severely from sciatica, which may have been "lightning-pains ;" another was subject to "rheumatic achings in the limbs," and liable to a nervous cough, which sometimes produced vomiting, had had severe sick headaches, and an attack of most violent neuralgia in the face, lasting a few days ; a third had complained of occasional stabbing pains in the lower limbs, chiefly at night, and of constipation and flatulence.

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[1] "Medico-Chirurgical Transactions," vol. lxxi., p. 215. London, 1878.

An examination of the knee-jerk should never be omitted in any future examination of such cases, and would at once settle this question, which is as yet undecided.

Hutchinson is of opinion that this group of symptoms is due to disease of the lenticular or ophthalmic ganglion, which supplies the iris and the ciliary muscle, and that when this triad of symptoms is present, and there are no others, the seat of the disease can be in no other structure than the ganglion itself. It is, however, to be noted that in one of his cases (No. 5) the superior, inferior, and internal recti were weakened, while in another (No. 8) all the ocular muscles were paralysed. This state of things would at once carry the seat of the disease further backwards to a more central point, viz., the nucleus of the third nerve. Moreover in all these cases a specific treatment was instituted which, although it did not cure the symptoms which were present, yet may have prevented the disease from extending further in the nucleus of the third nerve. It is also difficult to explain, on the theory that the disease is in the lenticular ganglion, why the iridoplegia should precede the loss of accommodation, and why the ciliary muscle and the sphincter of the pupil should suffer in different degrees ; while this would be easily accounted for if we assume the seat of the disease to be in the nucleus of the third nerve. Another objection to Hutchinson's theory of the lenticular ganglion being in fault is that in the majority of his cases both eyes should have been affected, for it must appear highly improbable that there should be simultaneous disease of both lenticular ganglia, which are so far apart anatomically ; and it would seem to be much more in consonance with general pathological principles to assume that the disease is situated more centrally, and at a part where the centres for the muscles of the eye are close together.

In this respect recent researches by Hensen and Voelcker,[1] are of considerable interest. They found that there is in the dog a nuclear area in the posterior portion of the floor of the third ventricle and the aqueduct of Sylvius, which has definite relations to all the movements of the eyes. Stimulation of that portion of the area which is most in front, showed it to be connected with the ciliary muscle for accommodation ; the one next behind with the iris ; that portion which is between the third ventricle and the aqueduct, with the rectus internus ; while still further behind were found the centres for the rectus superior, the levator palpebræ, the rectus inferior ; and most backward that for the obliquus inferior.

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[1] Erlenmeyer's "Centralblatt für Neurologie," February, 1881.

This arrangement of the nuclei of the third nerve is most probably the same in man ; for Kahler and Pick have found in two cases in which the function of the iris and accommodation had been normal, that the anterior portion of the area just described was healthy ; while in one of these where there had been paralysis of the rectus internus, the median part of the area was destroyed, and in another, where the rectus superior, the obliquus inferior, and the levator palpebræ had been paralysed, the posterior portion of the area was found sclerosed. This arrangement likewise accounts easily for the independent affection of the rectus internus, which has been occasionally observed, as there is a separate nuclear area for this muscle.

6. *Olfactory derangements.*—*Anosmia,* or loss of smell may occur from congenital absence of the olfactory nerve ; and is not infrequent in the aged, where sclerosis of the nerve is sometimes discovered after death, affecting chiefly the external root, which may be traced to the fissure of Sylvius, and which seems to be more important for olfaction than the middle or internal root. In tabes anosmia may be owing to acute inflammation of the olfactory nerve, but occasionally it comes on more slowly, and is then no doubt owing to chronic degenerative atrophy of the same.

Case 52.—In October, 1878, a banker's clerk, aged 48 married and father of two children, was admitted into the hospital under my care. He had been in tolerably good health until about eight years ago, when, apparently without any particular cause, he suddenly began to feel numbness in his feet, and lost the proper perception of the hardness of the ground. It seemed to him as if he was treading on india-rubber balls, or bales of cotton. About the same time he was startled by perceiving a strong smell of phosphorus, which overpowered all other accidental smells, and never left him at all for about six weeks. At the end of that period he noticed that he had entirely lost the sense of smell for odoriferous substances of any description. The smell of phosphorus had then given way to a persistent and not unpleasant kind of scent-sensation, which he compared to that of civet, very much softened down. This sensation continued for several years, but was now likewise gone; and there was at present a total absence of olfactory sensibility. I tested the patient with assafœtida, ether, valerian, camphor, millefleurs, opoponax, and a variety of other strongly-smelling substances, either pleasant or disagreeable, none of which, however, produced the slightest effect upon his nose. He did perceive ammonia, which caused lachrymation and a choking sensation, just as in healthy persons, and also the vapour of strong acetic acid, and snuff, which caused sneezing. All these last-named substances, however, act chiefly on the nerves of common sensation, viz., the nasal twigs of the ophthalmic branch of the fifth nerve, and the spheno-palatine ganglion, which were in their normal condition.

The perception of flavours in eating and drinking was likewise almost entirely gone. It is well known that the gustatory nerve only responds to four different kinds of sapid substances, viz., the saline, the acid, the bitter and the sweet; and that flavours are recognized by the olfactory, not by the gustatory nerve. In accordance with this I found that when the patient's eyes were bandaged so that he could not see what he was eating or drinking, he was unable to distinguish between stewed onions, apples, and turnips ; although he could tell roast beef from roast mutton. He did not perceive the flavour of port wine or claret, but felt the former hotter than the latter, saying it was gin or brandy, while claret appeared to him like vinegar and water. These sensations were evidently owing to impressions made on the lingual and palatine branches of the fifth nerve, and partook of common sensation rather than of special sense. It is a singular fact that, until experimented upon in this manner, the patient was not aware that he had lost the perception of flavours, but thought that he tasted everything quite as well as previously to having lost his smell—vision and memory evidently supplying in this instance the lost sense. Besides anosmia, symptoms of tabes existed in the upper and lower extremities, the bladder, rectum, and sexual organs, and the patient offered one of the most perfect types of sclerosis of the posterior columns of the spinal cord that could well be found.

In this case the anosmia was evidently not owing to any affection of the accessory mechanism of smell, such as inflammation of the Schneiderian membrane, ozæna, polypus, adhesion of the soft palate to the posterior wall of the pharynx, paralysis of the portio dura, etc. ; for the entire accessory mechanism of that sense was found to be in perfect order. Nor could it be attributed to disease of the olfactory centre in the brain, which Ferrier has shown to reside in the subiculum cornu ammonis. If there had been a lesion extensive enough to destroy both subicula, surely other symptoms, and more particularly entire loss of taste, must have been present. In their absence it seemed permitted to assume that the lesion was a peripheral one, and was seated where the olfactory ganglia lie closely together, at the base of the brain, on the cribriform plate of the ethmoid bone. The clinical signs showed at first a stage of sensory hyperæsthesia which lasted for six weeks, and then merged into complete anæsthesia. This corresponds closely to what I have observed in neuritis of the fifth nerve, where there is likewise a stage of hyperæsthesia, as evidenced by severe pains in the parts supplied by that nerve, and lasting for five or six weeks, after which complete anæsthesia of the face and scalp, and paralysis of the muscles of mastication, which are supplied by the small root of the nerve, set in.

Why should this patient have perceived a phosphorous smell during the first period of his illness? I believe I am in a position to affirm that the olfactory nerve responds to stimulation other than by special odoriferous substances, by perception of the smell of phosphorus. It is well known that the constant voltaic current has a peculiar action on the nerves of special sense, which answer to its passage by certain well-marked sensations. Thus, galvanisation of the optic nerve causes flashes of light, of the gustatory nerve a coppery taste in the mouth, of the auditory nerve a singing or hissing noise in the ear. It is easy to demonstrate these facts, because the nerves I have just mentioned are very sensitive to the influence of the voltaic current, and therefore respond to a slight power; while the olfactory will only give an answer when a very high power is used. A very powerful voltaic current, however, when applied to the nose or any other part of the face, causes such disagreeable sensations of pain, giddiness, sickness, together with dazzling flashes of light and loud noises in the head, that experimenters in general have been unwilling to bear the inconvenience of the procedure, or unable to analyse all the various sensations perceived at the time. The fact, therefore, that the olfactory nerve does respond to the voltaic current by the perception of a phosphorous smell was not established until some years ago, when I demonstrated it in a patient who suffered from bilateral anæsthesia of the fifth pair of cerebral nerves.[1] In that case a strong voltaic current could be borne without inconvenience, because the patient was insensible to a moderate power. The patient was in perfect health, except as far as the affection of the fifth nerve was concerned. His smell was keen ; and when a powerful current was directed to the mucous membrane of the nose, which was insensible to ordinary stimulation, the patient invariably, and without being questioned about it, said, "I smell phosphorus"; just as he mentioned that he saw flashes of light when the current was directed to his eyes. It is fair to assume that irritation of the olfactory nerve by hyperæmia and inflammation will likewise cause a smell of phosphorus, such as I have shown to follow voltaic irritation of the nerve, and just as in retinitis flashes of light are perceived by the patient. All these circumstances taken together appear to me to warrant the conclusion at which I have arrived, that the case just related was one of *acute olfactory neuritis*, ushered in by hyperæsthesia, and marked in its later stage by anæsthesia of that special sense.

The patient died of collapse some months after admission, and Dr. Ferrier, who was then my colleague at the hospital, made the autopsy. Sclerosis of the posterior columns of the cord, with some spinal meningitis, was discovered, and on opening the skull, the naked-eye appearances of neuritis of the first pair at the base of the brain were seen. The specimens were removed, and handed for microscopic examination to the late Dr. Lockhart Clarke, who was then likewise physician to the hospital. Unfortunately, that gentleman died soon afterwards, and I have not been able to trace the specimens, which is much to be regretted in a case so unusual.[2]

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[1] *Vide* my paper, "On the Physiology and Pathology of the Fifth Pair of Cerebral Nerves," in "Transactions of the Royal Medical and Chirurgical Society," vol. lii., p. 27. London, 1869.

[2] *Vide* my Paper, "On Neuritis and Peri-neuritis of some of the

Occasionally, anosmia is evanescent, like palsies of the rectus externus or internus muscles. If it comes on without any apparent cause, such as injury to, or over-stimulation of, the olfactory nerve, etc., it is a suspicious symptom; and an examination of the knee-jerk should on no account be neglected.

Hyperosmia also occurs occasionally in tabes, and shows this peculiarity, that only unpleasant smells are perceived. There is never an impression of nice scents or sweet-smelling flowers, but rather that of rotten eggs, or any other kind of abomination, fæcal matters, burning sulphur, or phosphorus. A similar hyperæsthesia of taste may be observed, so that everything that is eaten has a horrible or sickening flavour, as of mud or fæces, and nothing ever tastes "nice." These forms of hyperæsthesia are often premonitory symptoms of certain forms of mental derangement which are apt to occur in the tabid, and which will be subsequently considered.

7. *Amblyopia and Amaurosis.*—Tabes is frequently accompanied by atrophy of the optic nerves, the anatomical characters of which have already been described (p. 26). The most important information on this condition is that which is furnished by ophthalmoscopic examination; yet it should not be forgotten that in the beginning of the malady the optic disc does not show any pathological alteration, and that the amblyopia of tabes may, therefore, at that stage be confounded with that occurring in diabetes and chronic alcoholism. From these latter the optic atrophy of tabes may, however, be distinguished by its always commencing in one eye, and by one eye being, in the further course of the disease, always worse (or better) than the other, except where the patient has be-

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Cranial Nerves," in "Brain," part v., p. 10, April, 1879, and my Lecture, "On the Physiology and Pathology of the Olfactory Nerve," in "The Lancet," May 21, 1881.

come stone-blind, while in diabetes and alcoholism optic atrophy is at once bilateral. Wasting of the fibres of the optic nerve is accompanied by shrinking of their capillary vessels. The optic disc, therefore, appears pale and whitish; when examined by the ophthalmoscope, a grey colour is mingled with the pink, and as the atrophy becomes more marked, eventually a peculiar glistening bluish or greenish mottled or mother-of-pearl tint is seen. At the same time the edges of the disc appear sharply defined, showing a distinct line of demarcation from the neighbouring parts; and to this is added a more or less considerable degree of excavation, from the surface of the disc being depressed. This is found to be in exact proportion to the degree of atrophy which may be present. Where there is much wasting of nerve-tubes and only little overgrowth of neuroglia, the excavation is, therefore, much more marked than where the connective tissue is much increased. In some cases, indeed, the nerve, although profoundly altered in its structure, is very slightly reduced in size, with the result that the excavation is minimal. The arteries of the retina are generally small and attenuated, resembling thin threads, but this is not invariable, as in some cases the vessels seem to retain their normal calibre. The small vessels on the disc are wasted, or have altogether disappeared. The veins of the retina may appear quite normal, and if reduced in size, they are less so than the arteries.

From this description it will be seen that the ophthalmoscopic appearances in the optic atrophy of tabes are entirely different from those of optic neuritis, as habitually seen in cases of intracranial tumour, and where atrophy may eventually follow the inflammation. In the latter case there are always traces of effusion about the disc, the edges of which are not sharply cut, but blurred; and a somewhat indistinct zone of transition is seen between it and the fundus of the eye. The arteries are reduced in size, and the veins tortuous and dilated. Another difference between optic atrophy and optic neuritis is, that the former invariably commences in one eye, and that the other eye begins to suffer a few months afterwards, while optic neuritis is at once bilateral.

The mode of evolution of optic atrophy is in general slow and continuously progressive. There may in the beginning be headache, pains in the eye, and subjective luminous appearances denoting a degree of hyperæsthesia of the optic nerve; the patient then sees sparks of different colours, fireworks, or insects floating before his eyes, and may have the sensation of a foreign body, such as dust or small bits of coal in the eyes; or there may be hemi-anæsthesia of the face. At the same time vision becomes indistinct, and the objects appear veiled, or as in a fog. This latter symptom, however, may also occur from loss of accommodation by paralysis of the ciliary muscle. A more definite sign of commencing amaurosis is a restriction or *contraction of the field of vision*, which usually commences at the temporal side, and only very rarely on the nasal side. This contraction after a time proceeds either laterally towards the centre of the visual field, or it creeps along its periphery both upwards and downwards until it reaches the opposite side; and only after the whole periphery has been invaded, the contraction advances towards the centre. It affects first one eye, and after a variable interval the other, where it often takes exactly the same course as in the one first affected. According to Galezowski, it is characteristic for the syphilitic form of amaurosis, that the contraction of the visual field first invades the entire periphery, and only then advances towards the centre. I have, however, seen a case (p. 106) where the affection was undoubtedly owing to syphilis, and where the patient stated that a dark cloud seemed to have become settled at the top of the field of vision in the right eye, and that he was unable to distinguish with that eye anything above the eyebrow. On the other hand, Hardy has seen a case where the lower halves of the visual field in both eyes were blind, so that the patient could only distinguish the upper halves of objects. This affection, however, was transitory, like palsies of ocular muscles, and disappeared at the end of a fortnight. These two cases however, show that Galezowski's statement does not invariably apply.

Another important symptom is presently added in the shape of *scotomata*, or breaks in the continuity of the visual field, causing the patient to see dark irregular spots before his eyes. The degree to which these scotomata interfere with vision, depends upon their situation, and is particularly great if they sit in the visual axis, while it is much less where they invade the periphery.

Colour-blindness (Daltonism, dyschromatopsia, achromatopsia) is also very common at this stage. The patient is unable to distinguish red and green, both of them appearing grey or black, or of a dirty brown colour, while blue and yellow may be readily recognised. After a time the colour-blindness becomes complete, and the patient can only distinguish between white and black. Dislike to bright light is also common.

Unless an energetic specific treatment is instituted at the very beginning of these troubles, the course of the affection is progressive and ends in complete blindness. The time which elapses between the first beginning of the amblyopia and the total amaurosis, varies ordinarily from one to six years. I have now a patient under my care in whom the right eye began to show symptoms of atrophy of the optic nerve in 1872. Mercury and iodide of potassium were then given, under the influence of which the affection became stationary in the right eye, and has up to this time (May, 1884) spared the left, although some other symptoms of tabes have since then become developed.

Optic atrophy is not nearly as frequent as Argyll-Robertson's symptom, or palsies of individual ocular muscles. It seems to occur in one case out of about ten or twelve, and may be one of one first symptoms of the disease, or is only developed in the later stages of it. I have found it to be invariably combined with loss of the knee-jerk in the very beginning, and these two symptoms may be the only signs of tabes for years. Sooner or later, however, other symptoms of the principal malady supervene ; and in La Salpêtrière Charcot has noticed that most women who are admitted into that infirmary with optic atrophy become tabid after a longer or shorter space of time. Spinal myosis is frequently combined with the optic atrophy of tabes, while in other forms of this atrophy the pupil is generally enlarged.

8. *Derangements of the Auditory Nerve.*—I have already mentioned that of the two portions of the auditory nerve, the nerve of space, or vestibulary nerve, suffers more frequently in tabes than the cochleary or true auditory nerve. Occasionally, however, both portions of this nerve are affected together :—

Case 53.—A butcher, aged 32, married, and father of three children, was admitted into the hospital under my care in January, 1876. He had been in good health until the commencement of 1875, when he began to feel poorly, and suffered from a troublesome form of indigestion, with nausea and loss of appetite (gastric crises ?). In May of the same year he suddenly began to squint and see things double, evidently from paralysis of one of the ocular muscles, although it would be impossible to determine at present which one of them was affected. These latter symptoms lasted only for a few days and then left him ; but shortly afterwards he was affected with vertigo and a roaring noise in the head. There was also a feeling of sickness, but no vomiting. Within a few days the tinnitus increased considerably, and appeared to him like thunder, or as if there were explosions of gunpowder in his head ; at other times it resembled the ringing of bells and screaming of whistles. There was at no time any loss of consciousness. This severe form of tinnitus lasted for rather more than a month, during which time the hearing of the patient, which before then had been perfectly good, was *gradually* much diminished, and at the end of the period mentioned he found himself stone-deaf. By this time the vertigo had left him, but, on going about, he noticed that he did not walk as well as before, more particularly in the dark, and was apt to stumble, especially on turning round; and he felt the peculiar sensation as if walking on cotton or bladders. Pain of a character peculiar to tabes began to shoot through the lower extremities, more especially in the night and on exposure to wet or cold. The ataxy increased rapidly, in spite of medical treatment, so that he became completely helpless; and when he entered the hospital, he had already reached the third stage of the disease, in which not only the co-ordination of movements but also muscular power suffers.

The examination of the patient proved unusually troublesome, as he was stone-deaf, and all questions had therefore to be written down for him on a slate. He was found to be utterly insensible to the shrillest and loudest noise, such as that of a cab-whistle blown just behind him, as well as to the sounds of musical instruments. He could not hear a watch tick when it was applied to the external ears or the cranial bones around; nor did he perceive the sound of a tuning-fork applied to the vertex and to the teeth. On applying the constant voltaic current to the ears, however, a distinct sound was perceived on making with the cathode and breaking with the anode. This sound the patient likened to a "blowing" or "ringing" noise, and it appeared to continue for some seconds after the current had commenced and ceased to act. This was over and above the habitual tinnitus, which never left the patient, and which was now of a moderate kind, resembling the flowing of water. The patient spoke intelligibly, and although he could not hear himself speak, he had no deficient or altered intonation of the voice.

The physiognomical expression was peculiar. His features appeared in perfect repose, and unimpressionable, except when a question in writing was put to him. Having noticed a similarly statuesque expression in a case of anæsthesia of the fifth nerve, from loss of cutaneous and muscular sensibility, I carefully tested the sensibility all over the face, but found it perfectly normal ; and the total absence of physiognomical expression was therefore in this instance owing to the patient being as it were shut out from the world, and being indifferent to what went on around him.

I may here remark that some time later I had the advantage of Mr. Dalby's opinion on the state of the patient's ears. He confirmed my diagnosis of the nervous origin of the deafness, as he found the external and middle ear, including the Eustachian tube and the tympanum, perfectly healthy. The conduction of sound was good, but the perception of it absent, and he therefore thought the deafness due to change in the nervous structures, either in the labyrinth or intra-cranial.

The patient had not inherited any tendency to nervous affections such as paralysis, insanity, or neuralgia. He had always been a steady, hard-working man, not given to alcoholic or venereal excesses. He had never had syphilis or gonorrhœa. He never smoked. He had, however, in his trade, as a butcher, been obliged to go about a great deal in all kinds of weather, and in the small hours of the morning, and had lately had much anxiety about money matters.

There were no symptoms indicating cerebral disease ; the intellect, memory, and speech being quite normal, and all the cerebral nerves, with the only exception of the auditory, were in full functional activity.

The spine was not tender to pressure or percussion, nor was there any spontaneous pain in it; and the pain in the limbs was less marked than it had been some time ago. There was incomplete cutaneous anæsthesia from the waist downwards to the feet, and also incomplete muscular anæsthesia. Tickling the soles produced no reflex movements, and pinching the gastrocnemius and rectus femoris produced hardly any sensation. The muscles were flabby and somewhat wasted, but responded freely to the voltaic and faradic currents. The patient could not walk at all, except when supported by two persons, and even then he had the greatest difficulty in stepping out, the peculiar jerking gait of ataxy being perfectly discernible. The helplessness was so great that it verged on paralysis. He could only stand when supported by two sticks, and when he closed his eyes he reeled like a drunken man. Yet he could, when lying down or sitting on a chair, move his legs and feet tolerably well.

The sexual power had been gradually lost during the last six months, and the bladder and rectum likewise participated in the disease. There was great difficulty in passing water, the patient having to strain for fifteen or twenty minutes before he succeeded in voiding a few ounces of urine. Occasionally there was incontinence. The urine was habitually neutral, and contained a large excess of urea and phosphates, but no albumen or sugar. The bowels were confined, and when purgatives were administered they often acted so rapidly that the fæces were voided before the patient had time to reach the commode.

The upper extremities were unaffected, with the exception of a slight feeling of numbness in the third and little finger of the left hand. The heart and lungs were healthy. The appetite, however, was very bad, and digestion much impaired; the tongue was furred. There was tenderness in the right hypochondrium, and increased dulness in the region of the liver. The patient was considerably emaciated, and had a sallow and dyspeptic complexion.

I have thus given somewhat fuller details of this case than of many others, because the patient completely recovered under treatment from all symptoms of tabes, excepting the deafness, which remained unaltered.

The lesion, which in this case caused the vertigo and the deafness, I believe to have affected that part of the auditory nerve which is situated in the membranous labyrinth. The deafness could not be considered to arise from disease of Ferrier's auditory centre, in the superior-temporo-sphenoidal convolution of the hemispheres; for this, although the centre of hearing, has nothing to do with the equilibration of the body. Nor could the vertigo be owing to disease of the middle lobe of the cerebellum; for this, although the central organ of equilibration, has nothing to do with the sense of hearing. The disease must therefore have been seated in the auditory nerve itself, which presides over both hearing and equilibration, and destructive lesions of which will cause deafness as well as vertigo.

At what part of the anatomical distribution of the portio mollis was the disease located? Evidently not at its root in the medulla, because there it is contiguous with the sentient root of the fifth nerve, and there would, therefore, no doubt have been anæsthesia of the face, together with the deafness. A case of this latter kind has been described by Professor Moos.[1] Nor was it likely that the nerve-trunk was affected where it emerges from the lower border of the pons Varolii. I believe this portion of the nerve to have been healthy, because there was galvanic response on applying the voltaic current to the ear, and such response appears to be absent in destruction of the nerve-trunk. We are, therefore, led to the conclusion that the disease affected the labyrinthine expansion of the nerve, comprising its branch for the cochlea as well as for the vestibule.

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[1] "American Archives for Ophthalmology and Otology," vol. ii., p. 199.

The pathological lesion in the labyrinth was no doubt of an inflammatory character, as there was a period extending over rather more than a month in which there were evident signs of special hyperæsthesia of the labyrinthine expansion of the nerve, which were followed by special and permanent anæsthesia. These symptoms correspond very closely to those which I have observed in acute olfactory and trifacial neuritis. The *gradual* loss of hearing during the period just mentioned speaks against hæmorrhage in the labyrinth, in which deafness is more suddenly developed. The cord-affection which followed the attack of auditory neuritis was likewise of a more markedly inflammatory character than is usual in ataxy, as the symptoms became developed with far greater rapidity than is seen in the majority of cases.

Inflammation of the labyrinthine expansion of the auditory nerve I believe to be more common than is generally thought, and it has no doubt often been confounded with congestion or inflammation of the brain, or been put down to an attack of severe dyspepsia and congestion of the liver. The chief difference in the clinical symptoms of hæmorrhage and inflammation is that the symptoms are not so severe and sudden in their onset in the latter, that they continue for a more considerable time, and become gradually more fully developed.[1]

The two parts of the auditory nerve may, however, be separately affected. The *nerve of space* may suffer either in its terminal branches in the semicircular canals, or through sclerosis of its nucleus in the medulla oblongata or in its origin in the cerebellum. Vertigo is, then, the principal symptom, and this may be associated with deafness owing to other causes, such as thickening or perforation of the membrana tympani, or obstruction of the Eustachian tube, without the cochleary nerve being at all implicated.

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[1] *Vide* my Paper on this subject in "Brain," part v., p. 16. London, 1879.

Slight attacks of *vertigo* are not at all uncommon in the first stage of tabes. The patient experiences a sensation of fulness and swimming in the head, and feels as if everything were spinning around; he staggers, and would lose his balance unless supported. Sometimes there is a kind of impulse to fall in a certain direction, either forward or backward, or to one particular side (either right or left). This would correspond to the different functions of the three semicircular canals, and of the several portions of the middle lobe of the cerebellum, as determined by physiological experiment. Thus injury of the superior canal and of the anterior portion of the middle lobe of the cerebellum has been shown to cause a tendency to make a somersault forwards, and to move the head rapidly forwards and backwards; lesion of the external canal, and of the lateral lobes of the cerebellum, causes rapid oscillations of the head and eyes from one side to the other, and tendency to spin round; while, lastly, injury to the posterior canal and of the posterior portion of the middle lobe of the cerebellum leads to rapid movements of the head backwards and forwards, with tendency to take a somersault backwards. The diagnosis as regards localisation may, therefore, in such cases often be pushed to a degree of refinement which would in former times have been thought absolutely impossible.

Vertigo is often accompanied with clammy perspiration, sickness, vomiting, and pain in the head and nape of the neck. Sometimes the exciting cause seems to be a sudden movement of the head, either to the side or upwards, or stooping. Such attacks of giddiness may come on even while the patient is in bed; he then feels the bed move and turn from one side to the other, or himself sinking through the floor, or falling out of bed, or raised into the air, and he grasps the bedstead or mattress in order to steady himself.

If the *cochleary nerve* suffers, the symptoms are tinnitus and deafness. Tinnitus is often felt as a ringing of bells, singing of birds, buzzing of blue-bottles, screaming of steam-whistles, explosions of gunpowder, or the rattling of an express train. Such symptoms may continue for a few days, and then disappear. A patient who was sent to me by Dr. Pearce, of Leicester (Case *53a*), and who had been affected with tabes for some years, when staying at a cold, draughty house, where there was much gas, to which he was not accustomed, one night awoke with a frightful noise in his head as if he was in the underground railway. This lasted half an hour or more, and then turned to a buzzing, which continued next day. He had at the same time a scalded feeling in the cheek, eye and nose. When he spoke or anyone else spoke to him, it sounded to him as if he was in a diving-bell, and any external noise seemed to produce a curious sound in his head. In a few days all these symptoms had disappeared.

Deafness in tabes may be quite accidental, and owing to various affections of the parts which transmit the sounds; such as accumulation of wax in the meatus, subacute or chronic lesions of the membrana tympani, or the middle ear, an impervious condition of the Eustachian tube from previous inflammation, etc. In other cases, however, it is owing to disease of the nerve itself, and is then generally progressive in character, so that the patient eventually becomes stone-deaf. Marie and Walton,[1] who have endeavoured to ascertain the frequency with which various forms of deafness occur in the tabid, have found it, in seventeen cases out of twenty-four, in patients who were inmates of La Salpétrière; and Ormerod has seen it in five cases out of thirteen.

9. *Fifth-Nerve Troubles.*—I have already drawn attention to the lightning-pains which may appear in the sphere of the fifth nerve (p. 149). Apart from these, however,

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[1] "Revue de Medicine." Paris, January, 1883.

other symptoms may show this nerve to be implicated in the morbid process, which is then most probably located in the ganglionic cells of the nuclei of the trifacial nerve in the medulla oblongata.

Anæsthesia of the skin of the face and scalp, and of the mucous membranes supplied by the fifth nerve, may occur either in one or both sides. The taste may be lost, and the movements of the tongue may become so awkward that the morsels are not properly moved about in the mouth during mastication and insalivation. Although the tongue derives its motor power from the hypoglossus nerve, and may, therefore, not be paralysed, yet the suitable and appropriate way in which the tongue generally does its duty is interfered with. In fact, there is ataxy of the tongue. The muscles of mastication, which are supplied by the minor portion of the fifth nerve, may also be in a state of paresis. At the same time there may be hypersecretion of saliva and epiphora.

Pierret[1] has described the case of a patient in whom there had been symptoms of tabes for nearly three years, when the face began to twitch, and the internal branch of the palpebral nerve became extremely painful. The patient appeared to be always chewing; when swallowing, he was obliged to do this very gradually and with extreme care, as otherwise everything "went into the wrong throat." Speech was difficult, and articulation awkward; when the patient was told to put out his tongue there were odd and jerky movements about the mouth, and the tongue was tremulous. Three years later all the muscles of the eye were paralysed; the patient kept chewing constantly; the saliva ran away from the corner of the mouth; the jaws were so weak in their movements that the teeth did not meet on attempting to bite off a morsel, and the patient had the greatest difficulty in seizing anything placed in his mouth between the teeth.

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[1] "Essai sur les Symptômes," etc., p. 42.

In another case reported by the same observer (*l.c.*, p. 44) there was, apart from other symptoms of tabes, almost complete anæsthesia of the mucous membrane of the mouth and the tongue. The patient said he had lost his palate; condiments were not tasted, nor was tobacco. The tongue had become clumsy and awkward, and could not move a morsel in the mouth. There were lightning pains in the teeth on both sides. Mastication was difficult; the patient could not whistle. Speech was impaired, and the tongue was subject to incoordinated movements. The nasal fossæ were likewise somewhat anæsthetic, and the patient complained of a constant bad smell. He also suffered from tinnitus and some degree of deafness in the left ear.

Hemi-atrophy of the tongue is also occasionally seen quite in the beginning of tabes, and may affect either side of the organ; one half of it shows furrows and fissures, and is the seat of tremor from fibrillary contractions. It may be slightly deviated to the opposite side. It does not appear to cause much trouble in speaking, masticating or swallowing, and is generally associated with palsies of the ocular muscles and atrophy of certain sets of muscles in the extremities. This symptom has as yet only been seen in syphilitic subjects, and should therefore, when seen, excite suspicion of that dyscrasia. I believe it to be owing to sclerosis of certain trophic fibres in the course of the fifth nerve, as I have seen a considerable degree of atrophy in the whole organ in a patient who was affected with bilateral disease of the fifth pair, without any other morbid condition.

10. *The portio dura and the glosso-pharyngeal nerve.*— These nerves appear to be but rarely affected. I have already related the case of a patient (p. 119) who was under my care at the hospital in February, 1878, who had had syphilis when seventeen years of age, and afterwards double vision and Westphal's symptom. When

he came under my care, he had paralysis of the left portio dura in the first portion of the Fallopian canal, viz., facial palsy, loss of reflex excitability and of faradic contractility, and increased response of the facial muscles to the constant voltaic current; but no symptoms showing any implication of the chorda tympani, the stapedian nerve, or the ganglion geniculum. He had also paralysis of the left rectus externus of the eye. Two and a half years ago he had had a temporary attack of hemiplegia of the left side, from which he had completely recovered in three weeks This rendered the syphilitic nature of the nerve-lesion certain.

Ataxy of the facial muscles has also occasionally been observed. As long as the patient does not speak, and is not under the influence of emotions, nothing particular is noticed; but when he converses, and more especially when he gets excited in talking, the features are seen to work in all directions, without synergy or co-ordination, producing grimaces; and this ataxy may also be seen in the muscles of the tongue, soft palate, and larynx. Conversation may then become very fatiguing, and the speech affected. In such cases the hypoglossus nerve is most probably also implicated.

Symptoms on the part of the *glosso-pharyngeal nerve* appear to be rare; occasionally, however, the uvula has been seen to be deviated by paralysis of the azygos uvulæ, which is supplied by the glosso-pharyngeal nerve. There may be also anæsthesia of the pharyngeal mucous membrane, rendering deglutition difficult.

11. *Crises and other symptoms in the sphere of the pneumogastric nerve.*

a. *Laryngeal crises* were first shown to be a symptom of tabes by Féréol in 1868, and afterwards investigated chiefly by the French school of pathologists, such as Vulpian, Charcot, Krishaber, Demange, Lecocq, Cherchevsky, and Dreyfus-Brissac. These symptoms seem to be more frequent in France than elsewhere, and, singularly enough, the only two patients in whom I have observed them happened to be Frenchmen. They may be amongst the first symptoms of tabes, or appear only at a more advanced period of the disease; and they differ from other laryngeal affections by the circumstance that they are entirely spasmodic in character, and unconnected with any catarrh or other affection of the larynx, the windpipe, the bronchial tubes, or, indeed, of any portion of the respiratory organs.

Three different forms of these crises may be distinguished, according to the degree of severity which they assume :—

a. The *slight attacks* resemble ordinary whooping cough; there is a succession of short, dry, loud, convulsive expiratory efforts, followed by a long whistling or whooping inspiration; the face appears red and congested, even cyanotic; the patient is excited and anxious; there is no, or only very slight, expectoration, but a tickling or pricking sensation in the throat. Such fits of coughing may come on without any apparent cause, or in consequence of a sudden impression of cold, or from excitement or emotion, or through indigestion, and they last from ten to about ninety seconds. There may be forty or fifty such attacks in a day; and they are often wonderfully persisting, and rebellious to treatment. One of my patients in whom they occurred, and whom I had the opportunity of observing for two years consecutively, was rarely a day free from them, winter or summer.

β. *Attacks of medium severity* are marked by great dyspnœa, stridulous breathing, and a feeling of impending suffocation. The face and conjunctiva appear more congested and cyanotic; the eyes protrude; the patient gasps for breath, and is on the point of choking. There may be epileptiform symptoms, such as biting of tongue, convulsions in the limbs, and involuntary evacuation of the urine. Consciousness, however, is not lost; on the contrary, the patient has the most fearful sensations of impending death by strangling or suffocation. There are headache, vertigo, and vomiting. Such an attack may come on suddenly during sleep, reach at once its maximum of severity, and last from five to ten minutes, after which all the symptoms vanish at once, leaving the patient breathless and exhausted, but otherwise well.

γ. The worst attacks are those of actual *apnœa*. The laryngeal spasm is so violent that the glottis seems to be quite closed, and respiration completely arrested. The patient falls down as in a fit, is unconscious, and may be convulsed or not. The symptoms therefore resemble those of epilepsy, or of the severest form of apoplexy. The patient may die in the fit, the heart's action being likewise presently arrested. Krishaber has in such a case performed tracheotomy, and thereby probably saved the life of his patient. After the operation, the crises became subdued, the laryngeal spasm being much less severe. The patient no longer lost his consciousness in such fits, and seemed to have rather more spasm in the diaphragm than in the larynx. This man wore a canula for some years afterwards, and, when he felt an attack coming on, opened it at once in order to allow air to enter the windpipe. Attacks of this latter kind may come on quite suddenly, or be preceded by a kind of laryngeal aura, such as a pricking, burning, or scratching sensation in the throat. Sudden draughts of cold air, or prolonged exposure to cold, appear to be the exciting causes. They may come on several times a day, and then suddenly disappear for months or years. They last longer than the less severe attacks, viz., from twenty minutes to two or three hours.

The laryngoscopic examination of the throat and windpipe yields, as a rule, completely negative results as far as any structural alterations of these parts are concerned. There appears to be, however, in general a hyperæsthesia of the laryngeal mucous membrane, and consequently undue reflex excitability. This is, no doubt, owing to sclerotic irritation of the nucleus of the vago-accessory nerve in the medulla oblongata. In a case of this kind, Cruveilhier found wasting of the roots of this nerve, as well of the posterior pyramids and softening in the corpora restiformia. Cherchevsky[1] has noticed laryngeal crises to precede the outbreak of other symptoms of tabes for thirteen years, just as optic atrophy may precede the evolution of other signs of the disease for a considerable time. He does not state whether there is loss of knee-jerk on the first occurrence of such laryngeal crises ; but such is most probably the case. Laryngeal crises must therefore be looked upon not simply as coincidences, but as actual legitimate symptoms of tabes, which owe their origin to sclerotic irritation of the nuclei of the laryngeal branches of the pneumogastric nerve, causing more or less violent spasm in the adductors of the vocal cords.

b. *Paralysis of the Abductors of the Vocal Cords* is another symptom which is occasionally observed in tabes. Dreschfeld[2] has recorded a case in which the laryngoscope showed paralysis of these muscles on inspiration, while the adductors acted normally. The patient's voice was unchanged and clear ; but he experienced slight dyspnœa on walking, and during sleep his breathing was accompanied by a loud stridor.

Felix Semon[3] has shown that, where there is central or peripheral, acute or chronic organic disease or injury of the centres or trunks of the motor nerves of the larynx, there is always either isolated paralysis of the abductors of the vocal cords, or that at least their paralysis is developed at an earlier period, and more complete, than that of the adductors, always supposing that there is not absolute transverse division of the parts, and therefore not complete paralysis, and that the cause is one of gradual invasion and progression. It appears that not a single case has been recorded by any laryngoscopic observer where primary structural disease of, or injury to, the brain or the nerve-trunks had given rise to isolated paralysis of the adductors of the glottis. On the other hand, in all functional affections of the motor laryngeal nerves, more especially in hysterical aphonia, there appears to be a peculiar tendency to paralysis of the adductors, while paralysis of the abductors under such circumstances is an event of the most exceptional occurrence. Possibly, even where cases of the latter kind have occurred, they have after all been such of structural rather than functional affection. Thus Buzzard thinks it probable that a case observed by Mackenzie and Semon, in which there had also been temporary ocular paralysis, was really a case of tabes ; but the case appears incomplete, as the knee-jerk had not been examined. Rosenbach, and after him Semon, have drawn attention to the analogy which exists between the tendency of the extensors and abductors of the limbs to be affected with preference, rather than the flexor and adductors, in organic disease of the nervous centres.

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[1] "Revue de Médecine," Paris, July, 1881.

[2] "Medical Times and Gazette," Sept. 17, 1884.

[3] "Berliner klinische Wochenschrift," No. 46, 1883.

It appears from this that if a vocal cord be found immovable in the position of phonation, and if there be no disease of the crico-arytænoid articulation or some myopathic process, this would lead to the suspicion of disease between the nucleus of the vago-accessory and the peripheral branches of the recurrent nerve, while inspiratory position of the paralysed vocal cord or cords renders it probable that the disease is local or functional.

Unilateral abductor paralysis does not cause any symptoms either concerning respiration or phonation. If only one crico-arytænoid posticus muscle is paralysed, the corresponding cord at first assumes the cadaverous position, but after a time a contracture of the antagonists is caused, and the cord is drawn inwards, and eventually becomes completely fixed in the median position. For this reason tranquil respiration in the adult remains unimpaired. It is true that during emotional excitement, efforts, etc., dyspnœa may appear, but it is slight, and probably more owing to the complaint which really causes the palsy. The latter can, therefore, only be recognised by a laryngoscopic examination, which is thus shown to be a very important aid in diseases of the nervous centres as well as of the thoracic viscera, in which the nuclei or fibres of the recurrent nerve may become affected.

c. *Paresis or paralysis of the pharyngeal branches* of the pneumogastric nerve may lead to difficulty of swallowing and regurgitation of liquids through the nose ; and when this occurs together with weakness in the legs, it may be mistaken for diphtheritic paralysis. In other cases there is from time to time a *spasm in the gullet*, which renders swallowing impossible ; and this may be so severe as to amount to actual pharyngism or œsophagism, and be accompanied with severe pain.

d. *Gastric crises* constitute one of the most singular and important symptoms of tabes spinalis. They may be, if not actually the first, yet one of the first signs of that protean disease ; and as cases of this kind have in former times been frequently confounded with congestion of the liver, ulcer, or cancer of the stomach, gout in the stomach, or believed to be owing to hysteria, lead-poisoning, or nephritic colic, or to cancer of the womb, ileus, volvulus, &c., Dolamore's[1] discovery that they may arise in the tabid, and are a special symptom of this malady, constitutes an important progress in diagnosis altogether.

The principal symptoms of gastric crises are pain and vomiting ; and these may either occur together, or there may be only one of these signs. The pain which is experienced, more especially in the hypogastrium, is truly agonising, and may radiate from the pit of the stomach to the chest, abdomen, and the os pubis. It may resemble a scald, or take the various other characters of lightning-pains (p. 146). It is often so excessive that the patient keeps on yelling at the top of his voice for hours, and eventually faints away, and remains for a considerable time in a state of syncope. A case is mentioned by Vulpian where a man, under the influence of this pain, knocked one thigh so violently against the other, that he broke the bone; and squeezed his arm so forcibly that he brought on paralysis of the muscles supplied by the musculo-spiral nerve, through compression of the latter. Such pain may continue for a whole week incessantly, and be hardly relieved by the most powerful sedatives, when all of a sudden it disappears as if by enchantment, and the patient feels that the attack is over.

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[1] "Des troubles gastriques dans l'ataxie locomotrice." Thèse de Paris, 1868.

Vomiting of food, mucus, bile, and coffee-ground matter, containing blood, is often superadded to it; but it may be the only symptom of the crisis. The vomiting may also be incessant, so that everything that is taken, either in the shape of food or medicine, is brought up as soon as swallowed, and the patient ultimately refuses to take anything at all. After the stomach has been completely emptied, vomiting and efforts at vomiting nevertheless continue, and then cause even greater exhaustion. Spasmodic singultus, eructations, cough, and meteorism are added, and the patient sometimes has a sensation as if his stomach were forcibly torn from his body. All this may last a few days, but it may also go on for months, causing great emaciation, and leading ultimately to death by inanition. The vomiting may, like the pain, also disappear suddenly, and apparently without being influenced by treatment. Such crises may occur every few weeks, and are then generally put down to ulcer or malignant disease of the stomach, more especially when the vomit is of the coffee-ground character. Blood may also be passed by the bowels and with the urine, and, in women, from the womb. During the crisis the pulse generally becomes very slow, falling occasionally to twenty-eight beats in the minute.

Case 54.—In October, 1882, the secretary of the Amalgamated Society of Carpenters and Joiners requested me to see one of their members, who had for two years been unable to work, and now claimed the benefit which that society allows to its disabled members. This man, who was thirty years old, but looked at least ten years older, denied having ever had any syphilitic infection, or committed any excesses in drinking, smoking, or sexual indulgence, but attributed his illness to prolonged exposure to cold, about two years ago. The first symptoms were violent attacks of vomiting, with great pain in the stomach, which came on from time to time quite suddenly, without any apparent cause. He would bring up first mucus, then any food which he might have taken before, and eventually bile. Such an attack lasted generally about twelve hours, during which vomiting and retching would be incessant, and not relieved by any medicine. At the expiration of the time mentioned, the symptoms would cease as suddenly as they came. A few months after the first of these attacks, the patient was seized with double vision, which, however, left him after a time. He then began to suffer from lightning-pains in the extremities, more especially the left arm and leg. At present there was a considerable amount of anæsthesia and analgesia, chiefly in the left side; loss of knee-jerk on both sides; ataxy of gait and Romberg's symptom; a wasted and flabby condition of the muscles; incontinence of urine, constipation of the bowels, and loss of sexual desire and power, rendering the diagnosis of tabes certain.

Gastric crises occasionally alternate with attacks of lightning-pains, so that a patient has hardly recovered from the former, when he is seized by the latter, whereby his life is rendered truly intolerable. Even if the vomiting be not prolonged, attacks of this kind leave their mark on the patient's constitution, which is afterwards much less able to resist injurious influences and more liable to give way under a slighter strain.

Whether these symptoms are owing entirely to derangement of the pneumogastric, or also of the sympathetic system of nerves, is at present not yet determined. It has been assumed that the phenomena of pain and spasm are due to irritation of the pneumogastric and the intercostal and spinal nerves associated with it, while such sufferings as faintness, distension, palpitation, flatulence, retching and vomiting are owing to the sympathetic nerve-disturbance. The lowering of the rate of pulsation which so often accompanies these crises, however, would certainly seem to point to the pneumogastric as the principal nerve involved.

e. Cardiac Symptoms.—The pulse in tabes ranges habitually from 100 to 120 beats, and even more, showing loss of power in the cardiac branches of the pneumogastric nerve, which is the regulator of the heart's action. The pulse is also frequently small and compressible. Leyden has seen two cases of *cardiac crises,* with dyspnœa and irregular pulse. During severe attacks of lightning pains, and gastric or intestinal crises, the rate of pulsation is apt to become very slow.

12. *The Spinal Accessory Nerve* does not seem to be very frequently affected. In July, 1878, however, I had a patient, aged 45, under my care, at the hospital, who had had syphilis twelve years ago, and in whom tabes began with lightning-pains in the shoulders and arms, and *torticollis* from spasm of the left trapezius muscle. This latter lasted for about three months, and then disappeared, together with improvement in the other symptoms.

13. *Early Cerebral troubles.*—a. *Aphasia and Paralysis.*—Attacks of aphasia, monoplegia, and hemiplegia, with or without loss of consciousness, are by no means uncommon in the first stage of tabes. They are often ushered in by a feeling of giddiness, which gradually merges into coma;

and paralysis of one or several limbs may then be discovered. These cases look at first sight like cases of cerebral hæmorrhage or embolism of an important artery in the brain; but as recovery takes place in two or three days, or at most in two or three weeks, it is evident that they are not owing to any gross organic lesions.

The pathology of these cases is obscure, the most probable supposition being that they are owing to some disturbance in the vaso-motor system of nerves. This may be spasm, causing ischæmia and temporary failure of blood supply to those portions of the brain which are affected; or paralysis, causing excessive hyperæmia in the blood-vessels and increase of intra-vascular pressure. Similar attacks occur in alcoholism, senile dementia, insular sclerosis, and the first stage of general paralysis of the insane. An examination of the knee-jerk, which is probably always lost where these symptoms occur in the tabid, will often be necessary in order to settle the diagnosis.

Attacks of this kind appear to be sometimes connected with laryngeal crises and epileptiform seizures, and may possibly be occasionally owing to the former. In some cases it is impossible to distinguish a severe laryngeal crisis from an epileptic attack.

A case in which hemiplegia of the left side appeared to be the first symptom of tabes has already been mentioned (Case 29, p. 119). The patient, who was then only nineteen years of age, but had had syphilis at seventeen, recovered completely from the hemiplegia in three weeks. Sometimes, however, hemiplegia occurs at a time when the usual symptoms of tabes in its second stage are fully established.

Case 55.—In August, 1883, Dr. Schmitz, of Neuenahr, sent to me a steward, aged sixty-three, a widower with one child, who had for some time past been under treatment for diabetes, and had visited Neuenahr in order to drink the waters there for the cure of that affection.

Almost immediately on his arrival there, however, he had an attack of hemiplegia of the right side, with aphasia, which rendered the Spa treatment impossible. He recovered rapidly from the hemiplegia, and Dr. Schmitz then advised the patient to return home. When I saw him, I found the symptoms of ataxy fully developed, both in the upper and lower extremities. The patient was quite helpless, had Romberg's, Westphal's, and Argyll-Robertson's symptoms, obstinate constipation of the bowels, and incontinence of urine. There was no trace of the hemiplegia left. The patient was, however, so confused in his mind, and had so much difficulty in remembering things and expressing himself intelligibly, that no further history of his case could be elicited.

Where aphasia and hemiplegia appear in the latest stages of the disease, they are generally permanent, and produced by organic changes in the vascular supply of the brain, causing destructive softening of the cerebral matter. Debove[1] has related the case of a man, aged forty-eight, who had for a number of years suffered from tabes, and was suddenly seized by right hemiplegia and aphasia, which disappeared within a fortnight. Four years afterwards he had a similar attack, but the symptoms then persisted, and death ensued a few weeks afterwards. Apart from sclerosis of the posterior columns of the entire cord, there was also found an area of softening in the left half of the pons Varolii, with secondary descending degeneration ; but there was nothing to indicate the attack which had taken place four years before. In another case, that of a patient, aged fifty-eight, tabes had existed for twenty years when he came under observation. He had an attack of aphasia and right hemiplegia in the eighth year of the tabes ; and these phenomena eventually disappeared in about eighteen months. Seventeen years afterwards he was still alive, and although tabid, was not paralysed. Buzzard, Ballet and Bernhardt[1] have recorded analogous cases, and Westphal has seen a man brought into the hospital for hemiplegia, when the loss of the knee-jerk in the paralysed limb led him to make the diagnosis of tabes. It is, therefore, seen how important Westphal's symptom is for the diagnosis of such complications of the principal disease· Lecoq[2] who has collected a number of similar cases, has likewise come to the conclusion that attacks of apoplexy are integral symptoms of tabes, which may occur at every stage of the disease, either by themselves, or associated with laryngeal crises and epileptiform seizures.

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[1] "Progrès Médical," Nos. 52 and 53. Paris, 1881.

b. *Epileptic seizures* may likewise appear in the first stage of the malady, and assume the form of general convulsions, with loss of consciousness, or of *petit mal.* In some cases these attacks are apt to recur at more or less regular intervals, while in others there is only a history of a succession of fits on one particular occasion.

Case 56.—In January, 1880, Dr. Schulhof, of Brook-street, asked me to see with him a merchant, aged fifty-three, single, who had had syphilis badly six years ago. The secondary manifestations ceased after six months, and the patient believed himself to be well, when two years afterwards he was, without any warning, suddenly seized by an epileptic fit, in which he lost his consciousness, bit his tongue, foamed at the mouth, passed his water, and was generally convulsed for about five minutes. This fit was followed by several other similar ones on the same day. On no subsequent occasion had he had any other epileptiform seizures or cerebral symptoms of any description ; but, soon after the series of attacks which he had had in 1876, various symptoms of spinal disease began to make their appearance, and had become steadily worse when I saw the patient. He complained chiefly of incessant nocturnal emissions of semen, which occurred four nights out of five, and sometimes several times in one night. This trouble nearly drove him to desperation. He also had ataxy of gait, and most of the symptoms of the second stage of tabes.

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[1] "Archiv für Psychiatrie," vol. xiv., p. 142. Berlin, 1883.

[2] "Revue de Médecine." Paris, June, 1882.

c. *Failure of brain power and mental alteration* of some kind is not uncommon in the first stage of the disease. The patient, who has previously been cheerful, interested in his occupation, and affectionate to his family, becomes morose, taciturn, irritable, timid, and indifferent to his interests. Sometimes there is so much mental depression that he bursts out crying at the least provocation or without any cause at all. Suicidal ideas are rife, and are occasionally acted upon.

Case 57.—A merchant, aged fifty-three, married and father of four children, consulted me in December, 1881. He complained of intense depression of spirits, saying that he felt as if the grey matter of his brain did not act, and that if a new lining could be put into it, he would be better. He also felt very restless, slept badly, and had frontal headache. There was some amount of divergent strabismus in the left eye; and some months ago he had been much troubled with double vision. He had had syphilis about ten years ago, and also several attacks of obstinate gonorrhœa at various times. He had lost his sexual power, but had no trouble with his bladder or bowels. He suffered, however, much from frequent emissions of semen in his sleep. The knee-jerk was entirely lost on both sides, while the quadriceps femoris showed increased excitability to direct percussion. There were no other symptoms of tabes. I did not see the patient again until August, 1883, when he complained of nothing fresh, but appeared a prey to intense melancholia. Three weeks afterwards he died suddenly, and I was given to understand that he had committed suicide.

Case 58.—A contractor in a very large way of business, aged fifty-one, married but childless, consulted me in December, 1882. He denied having had syphilis, but had notoriously led a very rackety life. He complained of failure of brain power which had come on rather suddenly about six months ago. While formerly he directed with the greatest facility all the intricacies of enormous commercial transactions, and the most complicated matters appeared to him like child's-play, he had suddenly found himself unable to attend to his avocations, to collect his thoughts or even to compose a letter. He could simply do nothing, and would sit crying in his room all day long. He had been under the care of the heads of the profession in Paris, but received no benefit. Although the case appeared to be at first sight one of simple break-down of his mental faculties from over-exertion, the presence of Westphal's symptom which I ascertained during my examination of the patient, led me to enquire about symptoms of tabes. It then appeared that, at the beginning of his illness, he had increased sexual desire, which had, however, now been changed into complete anaphrodisia and impotency ; he also suffered from constipation of the bowels, and difficulty of urination ; and had occasionally had shooting pains in the lower extremities. These were the only positive symptoms of tabes which I could discover, but which, in connection with Westphal's symptom, left no doubt on my mind that the failure of brain power was only a symptom of a more general disease.

Cerebral troubles which occur habitually in the later periods of tabes, more especially general paralysis of the insane, will be subsequently considered.

14. *Early symptoms in the sphere of sensibility.*—Apart from the lightning-pains which have already been described (p. 146), other symptoms occur at an early stage of tabes in the sphere of the sentient nerves which are highly characteristic of the complaint, and often important in a diagnostic point of view. These are chiefly hyperæsthesia to touch or temperature, certain forms of paræsthesia in

different parts, and numbness in the feet and the peripheral branches of the ulnar nerve.

a. *Hyperæsthesia* may occur together with occasional attacks of lightning pains, or exist by itself. The chief feature of this condition as seen in tabes is, that simple touch of certain areas of the skin, instead of producing the ordinary feeling of contact, is unpleasant and painful. This is chiefly seen in the spine and its neighbourhood, but may also occur in the lower extremities, and is at a later period of the disease sometimes followed by anæsthesia in the same parts.

Case 59.—A solicitor, aged fifty-five, married and father of six children, consulted me in May, 1882. He stated that, with the exception of gonorrhœa early in life, he had never been ill until about eighteen months ago, when he began to suffer from soreness and irritable feelings about the spine, more especially between the shoulders, but also in the loins. There was so much tenderness there that he could not bear to be touched. The least pressure on the vertebræ, more especially in the lower dorsal and upper lumbar region, was most distressing; and slight percussion of any portion of the spine made him wince and call out. A touch with a cold substance was particularly disagreeable, while he did not mind so much anything hot. He disliked the rubbing of the clothes against the back, and could not bear to have his coat brushed when he had it on; he could not lean his back against a chair, and slept on his side rather than the back. Any application of ointments, embrocations, etc., for the relief of this hyperæsthesia was particularly trying to him, and had not done him any good. This hyperæsthesia was constant; but the patient also suffered from occasional attacks of shooting pains in the arms and legs, and from a sensation of excessive tightness round the upper portion of the chest. He never had a headache, and there were no symptoms on the part of the cranial nerves. The knee-jerk, however, was completely lost in both sides, and there was undue excitability of the vastus internus to direct percussion. He could walk fairly well, and the gait showed no trace of ataxy. He had, however, a difficulty in standing on one leg, and staggered when standing with his feet close together and his eyes shut. There was no anæsthesia anywhere. His digestion was fairly good; but he was apt to vomit in the morning if he had taken only a little more than his usual allowance of wine the evening before. The bladder was sluggish. After breakfast he had invariably a sudden involuntary discharge of a small quantity of urine—about two tablespoonfuls; but such incontinence did not occur again during the whole of the day, and he had never wetted the bed. The bowels were regular, and under perfect control. The sexual desire and power were gone; but he had occasionally seminal emissions in his sleep, which made him feel very wretched the day after. The patient was therefore evidently in the first stage of tabes; but the hyperæsthesia of the spine overshadowed all the other symptoms, and was the only thing for which he sought advice.

b. *Paræsthesia.*—Of all forms of perverted sensation which occur in tabes, the most frequent and diagnostically most important is a peculiar feeling of *tightness, constriction,* or *compression,* which, from being generally perceived right round the chest, is also often called *belt* or *girdle sensation.* The chest feels forcibly or, as some patients express themselves, "fiercely" compressed or constricted, as if a cord or rope were tightly drawn around it, or as if it were laced in stays of the tightest possible fit, or squeezed in a vice, a press, or a straight-waistcoat. This is one of the most uncomfortable sensations which may be imagined, and often bitterly complained of. It causes difficulty in breathing, and great oppression. When it has once taken hold of a patient, it rarely leaves him, and continues year after year. It is apt to vary in degree, and when particularly severe, may take the form of an attack of asthma, so that the patient is unable to lie in bed, but is obliged to sit up or lean forward. It is generally worse at night. It is also felt in the lower part of the abdomen and the lower extremities. In the case of a Parsee gentleman, aged forty (Case 60), who was sent to me by Dr. Handfield Jones, in December, 1866, this was the principal symptom complained of; and it was also felt in the legs and feet, which felt as if they were encased in excessively tight elastic stockings or bandages.

Müller, of Wiesbaden, explains this curious symptom by vasomotor spasm and consequent constriction of the arterioles of the posterior columns; but this explanation is not very satisfactory, as vasomotor spasm is rather connected with a feeling of coldness and chilliness than of tightness. There can be no question that it is owing to sclerotic irritation of the posterior root-fibres; but the exact way in which it is produced is as yet unknown.

In exceptional cases a sensation of tightness is also felt in the tongue, teeth, face, and scalp.

Other forms of paræsthesia are much less common. Occasionally patients feel as if drops of hot or cold water were falling on their limbs, or as if large pailfuls of iced water were poured over them, or as if they were knocked by the fist or a hammer. One complains of a sensation as if there were a tumour in the stomach; another, as if one of his legs did not belong to him, or detached itself from the body; or of a feeling of fulness or emptiness in different parts. Most of these sensations are temporary; they may last five or ten minutes, then disappear, and reappear at more or less prolonged intervals.

c. *Anæsthesia* is generally not very marked in the first stage of tabes. There may, however, be numbness, and a feeling as if the parts had gone asleep, with pins and needles; while actual loss of sensation is very frequent in the second period of the disease. Certain parts of the body are affected by preference, and none more so than the soles of the feet, so that the patient feels uncertain about the nature of the ground on which his feet rest or move ; he often cannot tell whether he is standing on a carpeted floor or a bare board ; has a sensation as if his boots were padded inside, or as if he were standing or walking in the snow or sand, or on cotton wool or india rubber, or as if he were sinking into the ground, or as if the latter rose under his feet. He is still able to feel the prick of a pin, and even a simple touch by the fingers in those parts ; but this sensation is more blunt than it is in health. Plantar anæsthesia may be looked upon as a symptom of transition, belonging to the pre-ataxic as well as to the ataxic period. Where it is marked, ataxy is tolerably sure to follow presently. The soles of the feet, when touching the ground, give us a sensation of the condition of the ground on which we walk or stand ; and if this sensation is absent or perverted, it is natural that walking should be difficult.

In the upper extremities the point of predilection is the sphere of the ulnar nerve, so that numbness is experienced in the third and fourth finger and the corresponding part of the hand and wrist. This may be bilateral, but is usually confined to one side, at a time when both lower extremities are already affected. It is chiefly inconvenient to musicians, and more especially to violin-players.

Case 61.—In March, 1874, Mr. Jeffery, of Worcester, asked me to see a merchant, aged forty-five, married and childless, who had exceeded considerably in sexual pleasures, and had had syphilis nine years ago. He had had secondary manifestations for about six months, after which they ceased. The patient remained well until about two years ago, when, apparently without any cause, he began to lose the control over the bladder, there being incontinence of urine by day as well as by night. At present he was obliged to use three napkins for the night, which were thoroughly saturated in the morning. At the same time that the bladder first began to give trouble, the patient began to feel numbness in, and loss of control over, the third and fourth fingers of the left hand, which felt as if he had a glove on ; and this annoyed him particularly, because his chief pleasure in life was playing the violin, and this was very considerably interfered with by the loss of sensation. The numbness after a time spread right up to the shoulder-blade, and in front to the cardiac region, and thus affected the left and afterwards the right leg. The legs now feel "as if the feet were cut off across the instep." He also had lightning-pains, more especially in the left leg, and sometimes of such awful severity as to keep him awake the whole night. The sexual power was lost, and the bowels constipated.

In some cases we find circumscribed areas of the skin in different parts of the body completely anæsthetic, while close by all the different forms of sensation are perfectly normal. Benedict considers such a symptom as a proof of the syphilitic nature of the disease ; and certainly all the patients in whom I have seen it had unmistakably suffered from constitutional syphilis.

Case 62.—In October, 1882, Dr. Möbius, of Leipzig, sent a merchant to me, who was thirty-seven years of age, married, and had had two children. He had had syphilis eight years ago, more especially in the throat ; and had been insufficiently treated. One of his children has had marked symptoms of congenital syphilis. Four years ago he began to suffer from lightning pains and numbness in the lower extremities, and the muscles became thin and flabby. There was a degree of amblyopia in both eyes, with ophthalmoscopic signs of optic atrophy. Westphal's and Romberg's symptoms were present. The patient walked with some difficulty, yet had only quite lately walked seven or eight miles at a stretch, after which he was, however, very much knocked up. There was habitual retention and occasional incontinence of the urine ; the sexual power was much diminished, and he felt very unwell after having had connexion. *There was an area of complete anæsthesia round the the left knee*, and spreading to the upper portion of the left leg ; while in the right knee and leg all the different kinds of sensibility were perfectly normal.

In some patients there is anæsthesia of the penis at an early stage of the malady, when sexual power is still preserved. They may therefore have connexion, but have no feeling of pleasure during the act. I have seen the urethra to participate in this condition, so that the introduction of the catheter was not perceived by the patient.

There may be areas of anæsthesia and hyperæsthesia of considerable extent in the same limb ; and more particularly where tingling and pins and needles are felt in the fingers and toes, a degree of anæsthesia is almost always present. A symptom no doubt intimately connected with this condition is that farado-cutaneous sensibility appears to be very much diminished at an early stage of the malady. In order to ascertain this a faradic soft wire brush is placed on the skin, and the minimal current-strength which will cause a sensation is determined, and then compared with healthy averages (Erb, Drosdoff).

15. *Early symptoms in the Motor Sphere.*—Loss of some degree of motor power is very common in the earlier stages of tabes. There is as yet no sign of ataxy, but a feeling of heaviness and debility in the legs, which indisposes the patients to active exercise. They can still walk apparently well, but their legs do not appear the same as before. There is a feeling of intense lassitude. They have a difficulty in going upstairs or uphill, get easily out of breath, and shun such exercise as going up ladders, or standing on ladders, or crossing narrow bridges, or dancing, or riding on horseback. This feeling of weakness varies from time to time, and occasionally goes off altogether for a period, but is apt to increase considerably after any particular

effort or expenditure of power. Standing also becomes difficult and fatiguing, and is therefore avoided; and even at this early period there is a considerable increase in the symptoms mentioned, when the patient is the dark, and deprived of the aid of his eyes. He finds his legs occasionally giving way suddenly, so that he falls to the ground; and this may occur with or without a shoot of lightning pain. There may also be local paralysis of certain groups of muscles; such as the adductors of the thigh, so that there is a difficulty or impossibility to approach the knees to one another.

This symptom of exhaustibility and lassitude is very constant, but has nevertheless not much diagnostic importance, because it also occurs in a variety of other nervous affections, more especially in neurasthenia. It must, however, excite suspicion of tabes, if it is constantly experienced. In neurasthenia there are greater variations in the degree of motor debility, while the complaint of numerous tabid patients is that they feel *always* exhausted, morning, noon and night, without any intervals whatever.

16. *Difficulties of the Bladder.*—a. *Sluggishness of the bladder* in expelling the urine is a very common symptom of the earlier stages of tabes. It is apparently owing rather to a certain degree of anæsthesia in the mucous membrane of the viscus than to primary loss of power in the detrusor muscle. The desire to urinate is not felt, and the patient, therefore, passes water only once or twice in the twenty-four hours, not because he feels the want, but because he thinks that it is the proper thing to do. In consequence of this the bladder often becomes greatly distended; and this distension, in its turn, leads to atony of the muscular coat of the viscus. The stream is very feeble, and often ceases before the bladder is half emptied. The pressure of the abdominal parietes is called in to aid the efforts of the bladder, and flatus and fæces are occasionally expelled at a time when the patient is not prepared for such

an occurrence. The trouble is much greater when he is under the influence of excitement or in a hurry, or when other people are present, as, for instance, in a railway urinal. Some cannot pass water in the natural way, except when there is a simultaneous action of the bowels; and the aid of the catheter is required after a time.

Case 63.—In June, 1883, I was consulted by a merchant, aged 54, married and childless, who had had a chancre, followed by ulceration in the mouth five years ago. In 1881, he first began to feel the vision in the left eye to grow dim; six months afterwards the right eye became similarly affected. At present he is totally blind in the left eye, while with the right he can just make out No. 15 (two-line English) of Jæger's test-types. There is achromatopsia; the bladder is so sluggish that the patient never feels any desire to empty it, and only voids the urine once in the twenty-four hours, because he considers it his duty to do so. Although this habit has gone on for several years, the urine showed no signs of decomposition; it contained, however, an excess of urea and a small quantity of sugar. There was no retention or incontinence of the urine, but the patient spent about fifteen minutes over the act. At first he had to press and strain for about five minutes without anything coming away, and then the urine dribbled away drop by drop.

b. *Incontinence of the urine* is likewise a frequent symptom in the first stage of tabes, and much more commonly met with than retention. It may be of a spasmodic or a paralytic character. If *spasmodic*, a small quantity of urine, varying from about a teaspoonful to two tablespoonfuls, is suddenly expelled, most frequently in the morning after breakfast, but also at other times, for instance, during sleep or on first awaking in the morning, or at any time of the day when the bladder is full, and a sudden movement or effort is made. This form of incontinence, which in some patients appears as regularly as clockwork at a stated hour of the day, is entirely different from the *paralytic* variety of incontinence. The latter is owing to a degree of anæsthesia of the mucous membrane of the bladder, which is apt to come on at a somewhat later stage. The patient then urinates without knowing it, or having commenced to urinate by an effort of volition has no consciousness of having finished, and therefore continues to urinate into his clothes. Total incontinence, where the urine dribbles away night and day, is rare in the first stage of tabes ; but symptoms of catarrh of the bladder, with ammoniacal decomposition of the urine, are even then not uncommon.

c. *Vesical crises* are amongst the most distressing phenomena of the disease. The patient may be awakened in the night by great pain and irresistible desire to pass his water ; there is, however, an impediment to its passage, and only a few drops are passed at a time by unheard-of efforts. The pain radiates from the bladder into the hypogastrium, the urethra, the perinæum, the groin, the testicles, and the inside of the thighs, and may be of agonising severity. Sometimes a small pellet of mucus is thrown out from the urethra, with relief to the pain ; but more frequently the pain continues in paroxysms for ten or twelve hours, while the single fit lasts for one or two minutes, after which there is a free interval of a quarter of an hour or half an hour, and then there is another fit. A crisis may return once a week or so. Blood is occasionally passed at the end of it, and the patient imagines that he must have hypertrophy or inflammation of the prostate or stone in the bladder. His doubts are only set at rest by a careful exploration of the viscus ; but catheterism and sounding have often a bad influence in these cases, and seem to lead to greater severity of suffering during the crises.

The following case was remarkable by the long duration of the attack, which lasted a week, and during which the patient had hardly any rest; and also by being complicated with gastric crises :—

Case 64.—In December, 1877, I was consulted by a merchant, aged thirty-two, married and father of five children. He denied having ever suffered from gonorrhœa or syphilis; but he had had rheumatic fever seven years ago, and has now disease of the aortic valves with hypertrophy of the left ventricle. He had masturbated as a boy, and had been subject to frequent nocturnal emissions. His chief complaint now was of "a horrid nervousness"; and when speaking of this to me, he suddenly burst out crying. There was great want of energy and application, and he easily became flurried on the slightest occasion. He had frequently suffered from what he called "rheumatic" pains in the joints and back, more especially in damp weather. These attacks have been followed by numbness, which was chiefly marked in the right side. The knee-jerk was lost in both sides. The patient was subject to attacks of "dyspepsia," with nausea, vomiting, and flatulence, and such severe pain that he had fainted away under their influence. More lately he had had attacks of irritability of the bladder, which came on without any apparent cause, lasted for a week, and then disappeared as suddenly as they had come. During such an attack he had, by day as well as by night, incessant calls to pass his urine, without being ever able to pass more than a few drops at a time.

17. *Intestinal troubles.*—a. *Constipation* of the bowels is habitually complained of by the tabid at an early stage of their malady. The muscular coat of the large bowel is sluggish, and there is great difficulty in obtaining a proper evacuation. Many of our most valued aperients refuse service, and even large purgative enemata often prove insufficient. In some cases there is anæsthesia of the rectal mucous membrane, so that the contact of the fæces or the introduction of the enema-tube is not perceived.

Great straining efforts may be required, and the desire to void the contents of the bowels often ceases when the patient is ready to satisfy it.

b. *Diarrhœa* sometimes alternates with constipation, and may come on suddenly, without griping pain in the abdomen or epigastrium, and apparently quite independently of the food which may have been taken. It may last a few hours or a few days, disappear suddenly, and return after a time. In the intervals the patients may feel quite well, have a clean tongue, and a good appetite. Occasionally attacks of diarrhœa coincide with or follow bouts of lightning pains. Where the diarrhœa is very persistent and very profuse, it may ultimately become of a choleraic character, or be complicated with hæmorrhage from the bowel; and in both cases a fatal result is to be feared.

c. *Intestinal crises* are much rarer than gastric crises, but equally painful and distressing. They also occur in sudden paroxysms, there being frightful pain in the inside, with tenesmus, constant desire to go to stool, spasmodic and painful contractions of the muscular coat of the bowel, and mucous, bilious, or serous diarrhœa. The patient appears like one in the last stage of cholera; the voice is extinct, the extremities cold, the urine suppressed, and even cramps are not wanting. Alimentation soon becomes difficult or impossible, and the patient may die of collapse.

A special form of the intestinal crisis is the *rectal* crisis. There is a kind of anal neuralgia, which may be more or less severe, with a peculiar feeling as if a foreign body were forcibly thrust high up into the rectum, or, as some patients express it, as if they were being impaled.

d. *Incontinence of the bowel* is rather less frequent than the analogous symptom in the bladder. It occurs, however, sometimes at an early stage, more especially when purgatives have been taken, or after an enema. Sometimes it comes on when the patient makes an effort to urinate, or on laughing, coughing, and sneezing. As a rule the discharge is slight, and in some cases it only occurs once or twice in the year.

18. *Sexual Troubles.*—a. *Impotence.* In most cases there is early failure of desire and of erectile power. Where erections occur, they are evanescent, occur on waking in the morning, and subside when the patient attempts to utilise them. Where connexion is still possible, the act is short, the ejaculation almost immediate, and there is little or no pleasure, but great prostration afterwards. Occasionally there is actual pain, or a feeling of irritation, instead of pleasure, at the time of ejaculation; while in some cases the sexual power continues, more or less unimpaired, well into the second stage of tabes.

Case 65.—In May, 1884, I was consulted by a gentleman, aged 35, married and father of five children, who had contracted syphilis in 1868. The secondary symptoms were mild; there was a slight squamous rash, ulcerated throat, and alopecia. These symptoms were treated with iodide of potassium, which he could then take in large doses. In 1872, while out in the East, he partially lost the sight of the right eye from optic atrophy. In 1875, thinking that he was free from the disease, he married. The first child was born with a syphilitic rash, and the wife soon afterwards showed specific symptoms. He now had a course of mercurial treatment. In 1876 he began to suffer from neuralgia in his legs, especially after hunting; and in 1879 numbness commenced in the legs, with unsteadiness in standing and walking. At that time it was first noticed that the knee-jerk was lost in both sides. At present he is not much worse than he was five years ago, although rather more unsteady in walking. He can walk half-a-mile or more with the aid of a stick, the gait not being very markedly ataxic; but the numbness in the legs and feet is very great. The bladder does not trouble him much, but he has always to make water before washing his hands; if he did not do so, he would wet his trousers. The bowels are fairly regular; but if he should take any purgative medicine, or if the bowels become relaxed from any other cause, he must obey the call at once, or there would be involuntary discharge. He also has frequently feelings of fulness in the rectum, which come on for a few minutes and then pass off. *The sexual desire is as good as ever*; his last child is only a few months old, and even she had a slight specific rash when she was born, showing that the patient had not yet got rid of the venereal poison.

b. *Satyriasis* is rare, but occasionally occurs quite in the beginning. Painful spasmodic erections occur frequently, when they are not wanted, at any time during the day, and are very persistent during sleep. The penis is of stony hardness, and the erection continues so long that it becomes intensely painful. The parts are in such a state of hyperæsthesia and undue excitability that the patient dreads to have a motion or to pass his urine. A sudden noise, the vibration caused by riding in a carriage, some slight exertion, or excitement of any kind will sometimes start the erection, and produce the feeling of an impending emission. The whole nervous system may then gradually be brought into a state of extreme hyperæsthesia and weakness, rendering the patient more or less incapable of mental as well as physical exertion. There is generally an aching pain in the loins and the testicles; incontinence of urine is commonly present; and gastric and vesical crises may add to the troubles of the patient, who is then in a truly deplorable condition. Occasionally, in order to free himself from such troubles, a man will under these circumstances accomplish connexion six or seven times in a couple of hours, and repeat such a performance day by day. It is then often believed that he is particularly well and strong; but this condition lasts rarely longer than a few months, and is then followed by complete anaphrodisia. Eisenmann has, however, seen a case where satyriasis continued more or less strongly for a space of thirty years.

In my experience patients in whom satyriasis has been an early symptom of tabes have had naturally strong sexual instincts, and been addicted to masturbation early in childhood, as well as later in life, and to indiscriminate sexual intercourse from an early age. The sexual passion seems to be the great ruler of their life; and when it has once led them into syphilis, tabes is apt to become developed.

c. *Nymphomania* in women appears to be rare. Sometimes, however, they appear, more especially during periods of lightning pains, subject to lustful sensations, and abundant secretion from the vulva, vagina and uterus. *Menstruation* is generally not much affected, but continues regular up to an advanced period of the disease.

d. *Spermatorrhœa* is not very common, and likewise occurs chiefly in persons who have exceeded much in venereal pleasures. They are liable to frequent nocturnal emissions, with or without lascivious dreams; and also to ejaculation of seminal and prostatic fluid in the daytime, with or without erections. The mental depression which is consecutive upon this state of things is quite out of proportion to the loss of substance which the patients experience, and is often considerably promoted by reading quack publications.

e. Finally *aspermatism* may occur in the first stage of tabes. This condition is generally accompanied by malnutrition or actual wasting of the testicles.

19. *Perforating Ulcer of the Foot.*—There are several varieties of this affection, which may be either a local disease or a symptom of a more general malady. Thus it may come on from suppuration of a bursa beneath a corn, when it may be supposed to be entirely local in its origin. Such cases may get perfectly well under surgical care, and

the patient may never develop other symptoms. Southam[1] has recorded a case where the patient had a perforating ulcer on the outer aspect of the phalangeal joint of the great toe, which had commenced with suppuration beneath a corn. Amputation of the toe was performed at the metatarso-phalangeal joint; the patient got well, and never had an ache or pain afterwards.

Perforating ulcer has also been seen after compression of, or injury to, the sciatic nerve, but is more especially found in connection with tabes, of which it may be the first or one of the first symptoms. In this latter case, it is almost invariably symmetrical in both feet; or, if there is an ulcer in only one foot, there is at least a hard callosity in the corresponding part of the other, which may in course of time develop into an ulcer. It is frequently followed, and sometimes preceded, by lightning-pains, gastric crises, and palsies of ocular muscles.

The following is an instance of perforating ulcer being apparently the first symptom of tabes:—

Case 66.—In May, 1884, the secretary of the Amalgamated Society of Carpenters and Joiners asked me to examine one of their members, who was thirty-eight years of age, married and father of two children. Four years ago, after having worked much in damp shops, and standing a good deal on the bare ground, this man found a painful soreness commencing in both soles of the feet, which gradually turned into perforating ulcer. He was admitted into the Middlesex Hospital, and gradually began to improve, but never got thoroughly well. Even now he has a deep circular sore, as large as a sixpence, in the centre of each sole, surrounded by an area of thickened skin, and complete anæsthesia and analgesia, while at the edge of the foot sensation is perfectly normal. In walking, he has lately had a sensation as if he walked on a carpet. About twelve months ago he noticed that he could not stand well when washing himself in the morning, and that he felt giddy, and had to look to his feet to prevent himself from falling. This trouble has increased of late. He now complains of tightness in the chest and difficulty in breathing. The knee-jerk is completely lost on both sides, but the excitability of the quadriceps femoris to direct percussion is not increased. Ataxy in the lower extremities is marked. He has had no symptoms on the part of the bladder, bowel, or sexual organs, nor higher up than the waist. He denied having ever suffered from syphilis, had had no accident, and never over-exerted himself in walking or otherwise.

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[1] "British Medical Journal," June 23, 1883.

It would be difficult to determine whether in this case the cause of the ulcers was peripheral neuritis, although the anæsthesia and analgesia round the sores made this probable. There could, however, be no doubt at all about the existence of tabes at the time when I examined the patient; and it would require a great stretch of imagination to suppose, with Page, that in such a case as this a corn led to the ulcer, and the ulcer to tabes. It is much more likely that the influence of wet and cold, by the patient standing much on the bare ground, led first to the spinal affection, and that the perforating ulcer was one of the first, if not actually *the* first, symptom of tabes; and that, in the natural evolution of the disease, the other symptoms which have just been mentioned made their appearance. Unfortunately, it is generally impossible in these cases to find out at what period of the illness the knee-jerk was lost; for practitioners and operating surgeons are still very far from having realised the immense practical value and diagnostic importance of this symptom.

In other cases, the ulcer may appear some time after definite symptoms of tabes have already shown themselves. Ball and Thibierge, Hanot, Treves, Fayard, Duplay, Morat, and many other surgeons have recorded examples of this affection. Such ulcers may heal, while at the same time the spinal disease may be progressing; but relapses are exceedingly common as soon as the patient leaves the horizontal position and returns to work.

20. *Arthropathies and other Trophic Affections.* — The peculiar joint-affection which Charcot was the first to describe (p. 35), may come on in the first stage of tabes, and may indeed be, if not the first, at least one of the first symptoms of it; but it is, perhaps, on the whole more frequent in the commencement of the second stage, when the symptoms of ataxy begin to become developed. Volkmann has endeavoured to account for the origin of this joint-affection by the ataxic movements of the patient producing shocks to the inner surfaces of the joints, and contusion of the ligaments and capsules; but this explanation is controverted by the simple fact that arthropathy may occur before any ataxy exists. It seems probable that arthropathy is owing to that form of peripheral neuritis which is known to accompany not unfrequently certain diseases of the centres of the nervous system.

Arthropathy is not uncommon, for Charcot has found it in one out of ten cases of tabes; and, since he directed attention to it, it has been not unfrequently seen by other observers. It seems to affect chiefly the large joints, more particularly the knee, shoulder, elbow, hip, and wrist, in the order in which they have just been mentioned, while the smaller joints of the carpus and metacarpus, and tarsus and metatarsus, etc., are more rarely involved.

The first symptom is generally a swelling, which may or may not be preceded by crepitation. Great rarefaction and atrophy of osseous tissue, and more especially of the articular ends of the bone, has, however, preceded this for a more or less considerable time. The initial crepitation is evidently caused by the separation of small pieces of bone, which break through the capsule, and thereby make a path for the synovia. This latter is effused into the cellular tissue of the limb, and thereby causes the swelling. The skin over the swelling is hard and resisting, and there is no pitting on pressure. The swelling may reach its maximum within a few hours, and loose bodies of the size of peas or filberts may float in the liquid. Dreschfeld[1] has found a mass of bone more than an inch long in the sheath of the sartorius muscle, and moving about freely with the contraction of the muscle. There is no pain, either spontaneous or on moving the limb, or manipulating it, showing absence of inflammation; and the only trouble which the patient experiences is caused by the mechanical distension of the limb through the swelling. There are no symptoms denoting any general disturbance of the system or any local reaction; but lightning-pains have often been found to precede the first symptoms of the arthropathy.

I have already mentioned that we distinguish between a benign and malignant arthropathy (p. 35). In the former the swelling may become dispersed in a short time, viz., from two to ten days; while in the latter the symptoms may continue for an indefinite period, and lead eventually to irreparable disorganisation of the joints, hypertrophy, or more frequently atrophy of the epiphyses, absorption of bone, and complete or incomplete dislocation.

If the hip-joint is affected, the head of the femur undergoes spontaneous dislocation. It leaves the cotyloid cavity, and enters the external iliac fossa, causing the leg to become three or four inches shorter. The head and neck of the thigh-bone are gradually entirely destroyed, the acetabulum is depressed and partly wasted, and the cartilages are likewise destroyed. The head of the femur may be felt as a distinct projection under the skin; it is freely movable and may be easily reduced, but soon slips out again from the acetabulum. If the knee suffers, it may be gradually dislocated backwards, so that the upper surface of the tibia is felt under the skin. The patient may at first still be able to go about, as the swelling is painless; then the leg suddenly gives way, the knee appears dislocated, and the patella is pushed inwards.

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[1] "The Lancet," July 10, 1880.

If the lesion is more in the shaft of the bone, spontaneous fracture may be the result, such as we see it in paralysed and insane persons, where there is abnormal fragility of the bones.

Arthropathy occurs not only in tabes, but after injury to peripheral nerves, where Weir Mitchell has seen it together with herpes, glossy skin, rapid muscular atrophy, etc. It is occasionally seen in hemiplegia from cerebral hæmorrhage or softening; and in other diseases of the cord, such as acute myelitis, tumour of the grey matter, paraplegia from Pott's disease, and hemiparaplegia from injury to the cord.

Nutritive disturbances, which are intimately connected with tabes, occur in other parts besides the joints. The *skin* is generally dry and cold, and subject to eruptions, such as herpes, pemphigus, and ichthyosis. Buzzard has recorded the case of a man who had more than two hundred attacks of *herpes* in the course of twenty years. Other observers have seen *pemphigus*, appearing in the legs, after attacks of lightning pain, the bullæ varying in size from that of a lentil to a crown piece, and fading away again in five or six days. Erythema, lichen, eczema, and urticaria are also occasionally seen. *Ecchymoses*, or small hæmorrhages in the skin are not infrequent. These effusions gradually undergo the usual changes, and disappear in about a week, without leaving any further traces. They are seen after painful attacks in those limbs which have particularly suffered. *Itching* is sometimes so violent that the patients dislike it as much as a gastric crisis. An eruption resembling *ichthyosis* has been seen by Ballet and Dutil, occupying nearly the whole body. It was slowly developed, and appeared chiefly in places which had previously been subject to anæsthesia, hyperæsthesia, or lightning pains. The skin was dry, thickened, livid, and in a state of constant desquamation. The upper extremities are particularly liable to this affection, and the back of the hand is sometimes so disfigured as to resemble pellagra.

The *nails* are also subject to malnutrition, especially those of the big toes. The nail first becomes black, as if it were suffused with blood, and after a time is found to be loose, and falls off. The matrix and skin in the neighbourhood may be anæsthetic or hyperæsthetic ; and there may be a dull, continuous aching in the toes, which is increased by pressure. The nail, however, falls off without suppuration, after which the pain is relieved ; and it seems to grow again quickly ; the reproduced nail may be normal or deformed, but appears mostly thickened, raised, crumpled, marked by longitudinal or transverse furrows, hard, and without its normal transparency. Sometimes the nail of the little toe is affected in a similar manner, but this kind of dystrophy is not seen in the finger-nails. Nails sometimes fall off but once a year ; and the same may occur in cases of insular sclerosis, and hemiplegia from vascular lesions of the brain.

The *teeth* may likewise fall out spontaneously, more especially where lightning-pains have occurred in the face, and where there have been laryngeal and gastric crises. In a case recorded by Demange, where this had occurred together with anæsthesia of the skin and the mucous membranes of the face, and with loss of taste, inspection showed some years afterwards all the nuclei on the floor of the fourth ventricle to be sclerosed, and the trunks of both fifth nerves contained numerous sclerosed fibres. In another case, where there had been anæsthesia in the sphere of the left fifth nerve, with falling-out of teeth from the left upper jaw, the autopsy showed sclerosis of both fifth nerves, the left being changed into a grey gelatinous thread, which was hardly recognisable. The Gasserian ganglion was reduced to a flat bit of connective tissue, and sclerosis of nerve-nuclei was found in the fourth ventricle.

The occurrence of *muscular atrophy*, owing to propagation of the disease to the anterior cornua of the spinal cord, has already been mentioned (p. 23); and cases have been given (pp. 106, 108) in which this was observed during life. With the exception of the muscular wasting, all the other forms of nutritive disturbance are probably owing to local nerve-lesions rather than to disease of any portion of the spinal cord itself.

21. *Vasomotor Symptoms.*—Signs that the vasomotor or sympathetic system of nerves is suffering in tabes are not wanting. A very common complaint of patients is a feeling of *cold* and chill, more especially in the feet and legs, which may be owing to spasm of the vasomotor system, or to loss of excitability in the vaso-dilator nerves. In such cases friction, or faradisation of the skin by a powerful current applied with a wire brush, does not cause any reddening of the skin, or at most a slight degree of it.

Hyperidrosis is occasionally seen in the palms of the hands and the soles of the feet, and is sometimes accompanied by seborrhœa of the scalp. Ebstein has seen unilateral sweating, with disease of the sympathetic ganglia on the affected side of the body only. This agrees with an observation of Bernard's, that after section of the sympathetic nerve in horses, there was sweating on the affected side, which he ascribed to temporary paralysis of the ganglionic cells. Remak has recorded a case of hyperidrosis in the face, head, and armpit on one side, which was increased if the patient partook of condiments, such as mustard, vinegar, etc.; there being also unilateral myosis, and increased temperature in the external meatus. On the other hand, there may be *anidrosis*, or total suppression of perspiration. Putnam[1] has described a case where a patient perspired in the upper portion of the body, but not anywhere below the umbilicus. Injection of five milligrammes of pilocarpine caused the upper part of the body to perspire profusely, while the lower portion remained quite dry. On another occasion the dose of pilocarpine was increased to a centigramme ; after which the upper part of the body perspired more profusely, the abdomen very slightly, the thighs just a little, while the legs and feet remained perfectly dry. In two other patients in the hospital, in whom the same dose was injected at the same time, perspiration in the lower limbs was quite as profuse as in the other parts.

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[1] "Recherches sur les troubles fonctionnels des nerfs vasomoteurs dans l'évolution du tabes sensitif." Paris, 1882.

The same observer has noticed *sialorrhœa* in two cases of tabes. There was suddenly during sleep an abundant flow of saliva, which in one patient ran from his mouth like a stream ; the attack lasted about a quarter of an hour, and the quantity of saliva discharged on that occasion amounted to about sixteen ounces.

Whether there is any *gastrorrhœa*, apart from gastric crises, does not seem quite certain. Occasionally no doubt, the vomiting of large quantities of water, amounting to ten or twelve quarts in a short time, is almost the only symptom of an attack ; but the sudden onset and the equally sudden cessation of it, apparently without being influenced by treatment, resemble very much what takes place in a gastric crisis.

The same considerations apply to attacks of *diarrhœa*, which have already been mentioned (p. 210), and which are probably owing to sudden loss of power in the inferior mesenteric plexus of the sympathetic system of nerves.

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Such are the symptoms which do or may occur in the first stage of tabes spinalis ; and it is impossible not to be struck by the extraordinary variety which they manifest. There is no other disease in the whole range of pathology which may commence in such extremely different ways as tabes; and it is undoubtedly this circumstance which accounts for the mistakes which have been and are even now so frequently made in the diagnosis of the earlier stages of that disease. Thus we may at the same time have a number of patients under our care who are all in the first stage of tabes, and who have yet hardly a single symptom in common, with the only exception of the loss of the knee-jerk. One of them may complain of loss of sexual power; another, of hyperæsthesia in the back; a third, of indigestion and a sensation of tightness round the stomach; a fourth, of rheumatic or neuralgic pains; a fifth, of failure of sight, and inability to lift the eyelid; a sixth of a feeling of intense lassitude in walking, and so on. This shows the great importance of being intimately acquainted with all the truly protean forms which this extraordinary malady may assume—an importance which is by no means only theoretical, but chiefly practical; for it is in the first stage of tabes that our therapeutical efforts are more likely to be crowned with success than at any later period in the evolution of the disease.

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II. *Symptoms of the Second or Ataxic Stage of Tabes Spinalis.*—The second stage of tabes is occasionally ushered in by fever. The patient feels chilly, and afterwards hot; the temperature rises to 100° or 101°; the pulse beats at 100 to 140; there is great lassitude, loss of appetite, a coated tongue, and constipation of the bowels. A feeling of fulness in the head, with vertigo, and sometimes tinnitus, is complained of; lightning pains, which may have been dormant for some time, are apt to revive, and considerable hyperæsthesia may exist in the back. Tingling and "pins and needles" are felt in the limbs; and there is often considerable mental depression, with a sense of impending danger. These symptoms generally last only two or three days, or at most a week; but if the patient is then again carefully examined, he is found to be in a worse condition than he was before the attack. Febrile movements of this kind may occur several times in the course of the disease, and always point to an increased activity of the morbid process, and mark another step in the downward career of the patient.

The principal new symptom which now appears, and which imparts an entirely new aspect to the malady, is that of ataxy. From this time forward, the ataxy overshadows more or less all the other symptoms of the disease, with the result that patients in the second stage of tabes resemble one another a great deal more than in the first. The extreme differences which we have seen to exist in the pre-ataxic stage begin now gradually to fade away, and the diagnosis is rendered proportionately easier.

22. *Ataxy.*[1]—The first who drew a distinction between ataxy and paralysis was Todd,[2] who stated that two kinds of paralysis might be noticed in the lower extremities,—the one consisting simply of the impairment or loss of voluntary motion, the other distinguished by a diminution or total absence of the power of co-ordinating movements. In the latter form, while considerable muscular power remained, the patient found great difficulty in walking, and the gait was so tottering and uncertain that his centre of gravity was easily displaced. In these few words we have a good description of the symptoms of ataxy or asynergy, as generally observed in the second stage of tabes spinalis. The term "ataxy" is as old as that of tabes, for both originate with Hippocrates, and both have entirely changed their meaning in the course of time. By tabes dorsalis the Father of Medicine understood a disease arising from sexual excesses, the chief symptoms being spermatorrhœa, marasmus, and hectic fever. The term ataxy, on the other

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[1] From τάξις, order, and privative Alpha (want of order).

[2] "Cyclopædia of Anatomy and Physiology," vol. iii., p. 721. London, 1847.

hand, was used somewhat indiscriminately for chorea, fevers, and various nervous disorders. At present, however, we understand by ataxy, not a disease of itself, but merely this special symptom, which consists of a want of co-ordination of complex voluntary movements, and a tendency on the part of the patient to lose his balance, but without actual loss of power, and apart from tremor, chorea, and paralysis. This symptom may be seen in disease of the cerebellum, and in alcoholic, saturnine and mercurial poisoning ; but it is more especially connected with that disease which has been long familiar to us as tabes. The first good clinical study of this symptom we owe to Duchenne, who gave a more minute analysis of what really constitutes co-ordination than any previous observer. He first drew a clear distinction between the different kinds of muscular action, viz., the executive and the controlling power ; and taught that in all voluntary movements, which we habitually carry out in life, there is not simply action, but also tempering and regulating of action by the antagonistic muscles. Without such mutual co-operation of different sets of muscles, movements would become devoid of certainty and precision, and would be more brisk and excessive than is necessary for the purpose which they intend to serve. Isolated muscular movements may be obtained by artificial means, such as faradisation, but do not occur in our common actions of every-day life. Complex muscular actions are learned early in life by incessant practice, and become in the adult automatic, so as to be carried out without an effort of volition. The loss of this slowly acquired faculty of co-ordination is therefore what we now understand by the term, ataxy.

Ataxy is apt to come on about four or five years after the first symptoms of tabes, such as palsies of ocular muscles, lightning pains, amblyopia, etc., etc., have made their appearance. In some cases, however, the ataxic stage is never reached, as the patients die previously of gastric and intestinal crises, collapse, bronchitis, and diarrhœa. In others, again, the pre-ataxic stage may last twenty years and longer before the second stage is entered upon. In the majority of cases the symptom of ataxy is slowly developed; but in a few it supervenes suddenly, more especially after over-exertion or severe exposure to cold.

In most cases which come under our care, we may distinguish three different epochs or periods of ataxy. Such a distinction has not hitherto been made, but I believe that it will be found to considerably facilitate the study and comprehension of the different aspects which this important symptom is apt to assume at different times in the progress of the disease. These periods I will characterise as follows:—

1st. *The initial period*, in which ataxy is so slightly marked that a skilled exploration is required in order to discover the symptom;

2nd. *The truly ataxic period*, in which the peculiar walk known as the "ataxic gait" is observed; and

3rd. *The period of muscular madness*, in which even the typical ataxic gait is no longer possible, and muscular action, as far as it still exists, is in absolute confusion.

A. *The Initial Period.*—It is difficult to say exactly when the ataxy of movements commences, as the transition from the first to the second period of the disease is often so slow and gradual as to be almost imperceptible. In some cases, however, it is noticed suddenly, when an unusual effort is made. The patient then finds that he cannot do what he wants to do, and begins to observe himself. In one of my patients (Case 21, p. 105) the ataxy was first perceived at a game of cricket, when he found that he could not run so well as before. Sometimes it comes upon people as a complete surprise, but they remember afterwards that somewhat similar occurrences have happened before, although at the time they did not realise it. Indeed, the muscular disorder may be at first so trifling that the patient succeeds by slight unconscious efforts in obviating any actual inconvenience.

At this initial period of the second stage of tabes, *a skilled objective exploration of the patient's condition is of paramount importance.* The subjective symptoms of which he most complains are often misleading, and comparatively insignificant; while the principal objective sign is often so concealed that only the specially trained observer is able to recognise and appreciate it. At this time a patient may still be able to walk four or five miles at a time without much fatigue, and often scouts the idea that there is anything wrong with his walking powers. It is, therefore, necessary to make him go through a certain number of tests, some or all of which, when ataxy is present, will infallibly reveal it. The more important of these tests are the following :—

a. Getting up.—A healthy person has no difficulty in getting up from a low chair or a couch, and in setting off to walk immediately afterwards. In the tabid, however, at this stage a certain amount of hesitation and awkwardness is noticed, when he is requested to get up suddenly and walk. He is seen to collect himself for an instant, then gets up, and waits a little while in order to balance himself properly, after which he sets off and then appears to have little or no difficulty in walking. The first steps, however, are often more awkward than the subsequent performance.

At this period both legs may be equally affected; but occasionally the difficulty appears to be in one leg only, or at least in one leg more than in the other.

b. Standing is likewise felt to be difficult. The patient dislikes it, and wants either to sit down directly, or to take hold of something to support himself. He sometimes feels giddy in standing, and instinctively prefers standing with his feet wide apart, so as to enlarge the centre of gravity as much as possible. This symptom becomes much more noticeable when the patient is requested to stand on one leg. Most healthy persons are able to do this quite well, at least for a minute, while the tabid then staggers about like a drunken man. He also has a difficulty in raising himself on his toes while standing.

c. A further sign at this stage is, that the patient is unable, when walking, to *stand still immediately* when requested to do so. Healthy persons have in general no difficulty whatever in doing this, although perhaps not quite as promptly as soldiers on parade, who are more efficiently trained for it. The tabid, however, shows an amount of embarrassment at such a juncture which is highly characteristic. If he attempts to stand still at once, he has to balance himself either by a forward or a backward movement, and sometimes manœuvres with his arms at the same time.

d. Another useful test at this period is to request the patient to *turn quickly round.* This act, although apparently simple, requires the harmonious action of numerous groups of muscles, and is generally wretchedly performed even at the commencement of the second stage of tabes.

e. A further excellent test, which I have not seen mentioned in any previous treatise on this disease, is *to make the patient walk backwards.* This faculty, which is chiefly practised and valued by courtiers, is nevertheless possessed by all ordinary mortals as long as they are in good health. For the tabid it is mostly very difficult to walk backwards at a time when he may have very little or no trouble in walking forwards. His heels seem to catch the ground; he dare not move for fear of falling; and if he succeed in walking backwards, it is in a peculiarly halting and odd fashion, which at once attracts attention. This symptom I have occasionally found before any of the other tests were available, and it is then, of course, of more particular importance.

I noticed this symptom for the first time in the case of a gentleman aged 42 (Case 67), who consulted me in February, 1882, and who was, in consequence of certain official duties, obliged to walk backwards a good deal. For years this had been the easiest thing in the world for him; but during the last six months he had been mortified to find that he experienced considerable difficulty in accomplishing this feat. He told me that his health was otherwise excellent, with the exception of "rheumatic pains" in the limbs, to which he had been subject off and on during the last three years. I examined him for the patellar tendon reflex, and found it absent in both sides. In the further course of the interview I elicited that the patient had had syphilis ten years ago; that he had a temporary attack of double vision about three years ago; that there was occasionally incontinence of urine in the morning and habitually a feeling of numbness in the soles of the feet. The patient was still able to walk exceedingly well in the daytime, and on a level road, but had found difficulty in walking in the dark. He went satisfactorily through the other tests which I am in the habit of using at this stage of the malady, showing that he was only just in the commencement of the second stage of tabes. The walking backward was indeed the most troublesome thing he had to contend with; and the contrast between the clumsy and awkward way in which he did this, and the apparent ease with which he walked forward, was indeed striking. When attempting to walk backwards, his heels seemed to become entangled in the carpet; he was evidently totally unable to raise his feet properly from the ground, and on one occasion would certainly have fallen unless I had supported him. All the muscles of the thighs and legs seemed to become rigid the instant he attempted to walk backwards, while in walking forward he appeared to have no difficulty whatever in bending his knees to the proper degree.

Since then I have made it a point to inquire about this symptom in patients suffering from tabes, and have found it present in the majority of cases. In an artist, who is now under my care, this difficulty is particularly annoying, because it prevents him from taking a perspective of his pictures by walking backwards from his easel. In this case there are no symptoms of tabes above the waist, so that the patient is able to paint as well as ever.

In difficult or doubtful cases therefore, more especially in those at the very threshold of the second or ataxic stage of tabes, the symptom which I have mentioned may put us on the right track, and lead us to examine the patient who shows it for other symptoms of the malady. As tabes is still frequently confounded with gout, rheumatism, neuralgia, dyspepsia, idiopathic amaurosis, and other conditions, any addition to our means of diagnosis for that time where the malady is not yet fully developed must be welcome.

f. The most striking test, however, at this period is "Romberg's symptom." Romberg[1] noticed, as far back as 1840, that the influence of light is a very important factor in such cases. He says that, even in the beginning, the patient must *see* his movements if they are to be at all certain. When at our request he stands up, and then shuts his eyes, he at once begins to stagger; and when he is in the dark, standing and walking become more awkward. A patient, who could see quite well, complained of being unable to work and dress himself in the dark, in the morning, without falling. Another man, who had to go to business at 6 a.m. in the winter, stated that he was obliged to have a servant to support him, which was not required in the daytime. At a later stage the patient may not even be able to sit on a chair when closing his eyes, but is apt to glide gradually down from it; and on coming from a well-lighted room into a dark one, he loses himself altogether.

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[1] "Lehrbuch der Nervenkrankheiten des Menschen," vol. iii., p. 184. Berlin, 1840.

Romberg's symptom is chiefly marked when the patient is requested to stand with his feet closely approached to one another, as then the point of gravity is more easily displaced. Even at an early period the patient begins to manœuvre with both arms, in order to prevent himself from falling. "He uses his eyes as crutches;" the reason for this being that the brain intervenes through the intermediate agency of the sense of sight, in order to preserve the balance of the body; and *he stammers with his feet.*

Some patients, when walking, look constantly at near objects, so as to be sure to find support on the shortest notice, while others are always looking at their legs and feet, and endeavour thereby to regulate their movements as much as possible. The manœuvring increases as the disease advances; but it may, in the beginning of the second stage, only be perceptible to the eye of the practised observer.

g. Finally, there is a peculiar symptom, which is not nearly so constant as those which have just been mentioned, but which nevertheless is of some importance. This is a disinclination on the part of the patient *to go downstairs,* while there may be no difficulty at all in going upstairs. To look down, more especially a long flight of stairs, appears to unnerve the tabid, who is anxious to secure the support of the baluster, and goes down very slowly, while looking all the time at his feet. The awkwardness of the performance is particularly noticeable when the observer stands at the bottom of the staircase. In a patient who was sent to me in January, 1880, by Mr. Hodgson, of Brighton, the first sign of ataxy was the dread which he experienced in going down ladders in the pursuit of his business as an architect, while he did not mind going up ladders at all.

B. *The Ataxic Gait.*—While, therefore, in the earlier stages ataxy has to be found out, and discovered by special modes of investigation, the phenomena which are observed in the further progress of the disease are so striking that no difficulty can be experienced in recognising them at first sight as part and parcel of the malady to which they actually belong.

After a time the difficulty in walking becomes so considerable that the patient is seriously alarmed by it. He finds it very troublesome indeed to get up from a chair, and has generally to make several separate efforts to do so ; he then stands with his body bent forward, his legs rigid and far apart, and with his arms stretched out, in order to increase the base of his support, and enlarge the centre of gravity. If we now examine his muscles, all of them feel completely rigid. The patient takes instinctively the greatest trouble to keep the ankle immovable, and to stretch the leg well on the thigh ; and he walks chiefly by the aid of the pelvic and femoral muscles. The knees are, therefore, not properly bent, so as to clear the ground well on going forward. While the paralytic is seen to have a difficulty to detach his feet from the ground, and often scrapes the boards or the carpet with his feet or boots, the ataxic, on the contrary, throws the feet too much forward and outwards, with a peculiarly rapid jerk ; the heel is therefore carried too far ahead to come down properly, is pulled back, and eventually comes down with a heavy thud or thump, stamping the ground. Sometimes the foot comes down flat, instead of on the heel, and the shock of this may be so great that it adds to the trouble, and the patient, in order to avoid it imparts to the leg a swinging motion. At other times he finds it impossible to walk slowly, but walks very fast, and actually runs without being able to stop or to change the direction in which he walks. Turning round is then extremely troublesome. All the time the patient is on his legs, he fixes an intense and anxious gaze on his feet, endeavouring to control their movements with his eyes to the utmost of his power, and so to prevent himself from falling, which is his constant dread. If at this stage he walks in the streets, and runs against people, or, what is more frequent, if people run against him, he is called "a drunken brute," and occasionally taken to the police station. He is presently obliged to walk with a stick, or to support himself on either side by a servant or a nurse.

C. *The stage of Muscular Madness.*—As the disease advances, the ataxic gait, as just described, loses its characteristic features. The patient is now no longer able to fix the ankle and knee joints, which used to be some check to the muscular incoordination of a former period. When he now attempts to walk, he searches continually with his feet for a support which he cannot find, his legs are utterly beyond his control, the movements altogether disorderly, and so many muscles act at the wrong time, and in the wrong combination, that no useful result can be obtained. There are sometimes real cramps, and such a degree of muscular madness that the feet fly about anywhere, and are knocked against the sticks or the legs of the persons supporting the patient. There is such kicking and sprawling that the patient gives the impression of a drunken man, or of one who is on skates for the first time, or who attempts to walk on board ship during a heavy gale. The efforts which he has to make to prevent himself from falling, by manœuvring at the same time with different groups of muscles over which he has lost all control, soon lead to exhaustion by the expenditure of so large an amount of muscular force; and the patient is, therefore, only too glad to go back to his couch, where he drops down with a sigh of relief, and in a state of prostration.

In some cases it is seen that the patient endeavours to check the muscular disorder by keeping the feet as close as possible to the ground, and therefore drags one leg after the other, so that the walk resembles at first sight that of paraplegia. It is however obvious, even at this stage, that there is plenty of muscular force left, as the patient may still stand erect when supported against a wall or by a heavy piece of furniture. Duchenne, in order to demonstrate this, used to raise himself on the shoulders of such patients when they were securely placed, and especially had their feet far apart, without their giving way ; and a dynamometer for measuring the force in the legs, which Messrs. Weiss and Son have constructed for me, has often at this stage shown me that the muscular power of the legs remained undiminished. The patient may also resist attempted flexion of the knee by calling into play the action of his extensors, or attempted extension by using the flexors ; and the simple stretching of the flexed leg, which he is still able to do at a very advanced period of the malady, is often performed with considerable force.

Crutches are sometimes of use to the patient. An army surgeon who was under my care in July, 1878, for a severe form of tabes, which, however, showed no symptoms above the waist, was absolutely helpless without crutches, but with their aid managed to walk about the streets of London, although in a sprawling fashion. In some cases, however, where the jerky and spasmodic element is very marked, crutches or even the support of other persons' arms are of very little use, as there is so much sprawling about with the legs that the crutches are thrown about in a useless fashion, and it may happen that an attendant, unless very steady on his own legs, is actually thrown over by the patient. Again, when there is ataxy in the upper extremities, the walking gets proportionately worse, since the use of sticks, crutches, attendants, and even taking hold of pieces of furniture, such as chairs, tables, etc., becomes impossible, and the patient is to all intents and purposes as completely helpless as one who is actually paralysed.

It is important at any time during the second stage of tabes to examine the patient in the *horizontal position*, when he is quietly lying on a bed or couch, as it is chiefly then that the immense difference which exists between ataxy and paralysis arrests attention. When a patient suffering from paraplegia, who is lying in bed or on a couch, is asked to move his lower limbs, the first thing he does is to help himself with his hands, and to make a general movement of the whole body, in order to bring the legs forward, or to change their position. The legs themselves remain almost or entirely inert, according to the degree of paralysis which may be present, while efforts are made by all the other parts of the body. In ataxy, on the contrary, even at an advanced stage, the patient has no difficulty in moving his legs in bed, although they may be quite useless for walking. There is only an impossibility of *orderly* and useful movements of the legs. The patient cannot raise the extended leg quietly and gradually into the air, as may be done without any difficulty by a healthy person ; on the contrary, when he attempts this, disorderly movements follow, he stretches the whole extremity, and throws it out of bed in a violent manner, either by a single bound or by several irregular jerks. The movement of abduction being particularly sudden and forcible, those who are near the bed may receive a kick before they have time to reflect. If you ask the patient why he stretches the whole limb whenever he is requested to make any movement, he replies that it is the only way to make any use of his muscles. He has also a difficulty in holding his leg up for more than a second or two, nor can he bring it down on the couch gradually. Again, if the hand of the observer be placed at some elevation above the bed or couch, and the patient be requested to place one of his feet in it, he can only succeed in doing so by chance ; in general there are ineffectual attempts made, in which the foot is jerked about in any direction but the one that was intended. If the patient be told to touch his own knee with the opposite heel, he may possibly be able to do it when looking on, but fails in accomplishing it with his eyes closed, at least for the first time, while after a few ineffectual efforts he sometimes succeeds in doing it. The influence of the eyes is, however, chiefly important when sensibility is at the same time much diminished ; and the power which is put forward for effecting a certain purpose is always in excess of that which would be used by a healthy person.

In the majority of cases the evolution of the ataxy is so slow as to be almost imperceptible. Occasionally, however, it comes on in a more acute form.

Case 68.—In August, 1881, Mr. Leftwich, of New Cross, asked me to see a male patient, aged thirty-six, who was employed as compositor at the Bank of England, and had been greatly overworked in the month of March. At the end of that month he was suddenly taken with lightning pains in the back and the left hip, inability to walk, and incontinence of urine. The patient strenuously denied any previous syphilitic infection. Romberg's and Westphal's symptoms, together with the other signs of tabes affecting the lower dorsal and upper lumbar portion of the cord were present when I examined him.

Cases of *acute ataxy*, like the preceding one, are most probably more of a functional than of a structural character. The patients generally improve a good deal under treatment in a short time, and sometimes get quite well.

Ataxy in the upper extremities is seen at an early stage in those cases where the disease commences in the cervical enlargement, instead of, as usual, in the dorso-lumbar portion of the cord. In most cases it begins in the upper extremities a considerable time after the lower limbs have become useless, and is then a sign that the morbid change is creeping upwards in the spinal cord. Here also, when ataxy is present, the patient may still have a powerful biceps, and squeeze the dynamometer almost to its utmost limits, showing that coarse muscular force is still present. In general we find that the ataxy is more liable to invade the muscles of the wrist and the fingers than those of the shoulders and the arm. The patient has a difficulty in buttoning his sleeves, in striking a match, in picking up small objects like a pin, in carrying a cup of tea to his mouth without spilling some, or in carving a joint. If we give him a pencil-case, or a knife, or a similar object, to hold in his fingers, he is apt to drop it. With his eyes closed, or in the dark, the awkwardness of his movements is still more marked. In France, a favourite test is to make the patient cross himself, and to notice the difficulty he experiences in making the sign of the cross with his finger. In this country a similar test is inapplicable in the majority of cases, and is replaced by the more secular one of requesting the patient to touch his nose with his first finger. It affords, occasionally, a strange sight to watch the peregrinations of the finger in search of the prominent feature. Patients who take snuff have a difficulty in indulging in their favourite pastime, as the snuff is often thrown to a considerable distance without reaching the nostrils, and occasionally loses its way into the mouth, where it is less acceptable. Carpenters, when still at work, let the hammer come down on their hands rather than on the nails they wish to fix; and skilled artisans, like watchmakers, jewellers, &c., are no longer able to carry out those minute and skilful manipulations which are required in their trades. The patient, after a time, becomes so clumsy and awkward with his fingers, and goes about his work in such a roundabout manner, that he ceases to be useful, and is discharged by his employers.

Musicians lose their facility in the execution of complicated passages which were formerly like child's play to them, and could be carried out without looking either at their fingers or their instrument. All manipulations with the fingers are apt to become more difficult when the patient closes his eyes. If one hand is put into a certain position, and the patient is requested to put the other hand into an analogous position, he is unable to do this without looking. When the ataxy reaches the stage of muscular madness, similar irregular movements take place in the upper, as we have seen them to occur in the lower extremities. Drummond[1] mentions the case of a patient who was searching for his left hand, having lost it under the bed-clothes, when suddenly the missing member flew up, and struck him a severe blow in the face. This arm often moved about by itself, being almost entirely beyond volitional control, and was even jerked about during sleep. These irregular movements appear to set in chiefly when parts of the skin, which are in a state of hyperæsthesia, are irritated.

The muscles of the body rarely show any ataxy. Respiration may, however, be irregular, and there may be swaying of the body and the head from one side to another, similar to nystagmus. The latter is habitually met with in Friedreich's disease, but not in tabes proper. When there is much ataxy in the muscles of the body, the patient is unable to sit on a chair or couch, and may even roll out of bed without being able to help himself. Difficulty in speaking, partaking of the nature of ataxy, is sometimes encountered; and the ocular and facial muscles occasionally show similar symptoms.

Ataxy of the ocular muscles was very conspicuous in the case of a patient, aged 41, single, who consulted me in June, 1884 (Case 69). He had had a chancre and bubo, followed by "plenty of secondaries" in the throat, ulcerations in the tongue, which had to be cauterised, and sores in the thigh. He underwent a long and persevering treatment, and seemed quite well, when he caught a severe cold in February, 1883, after which lightning-pains and ataxy of gait made their appearance. The pains felt partly like blows with a hammer, which were principally given on the knees; and sometimes as if thousands of fish-hooks had got hold of the calves of his legs, and were dragging them up. Romberg's, Westphal's, and Argyll-Robertson's symptoms are present. His legs are numb, and feel as if he had been asleep and lying in water for a long time. There are symptoms on the part of the bladder, bowels, and sexual organs which it is not necessary to detail. He always feels exhausted, and always in pain. There is numbness in both hands, more especially the left; and his back feels as if he had been struck by a thickly covered mallet, or as if he were suddenly being dragged down by the backbone. The most interesting symptom about him, however, was true ataxy of the ocular muscles. There was no paralysis in any of them, nor nystagmus; but the patient had the greatest difficulty in combining them to synergic action. The consequence was that he saw easily double, and that more especially a sudden purposive movement of the eyes was distressing to him. The most difficult thing for him was to look up to the ceiling, although the muscles concerned in this movement showed no trace of paresis or paralysis. He felt, however, so strained and giddy when attempting this movement, that he immediately afterwards closed his eyes, and threw himself back exhausted into his chair. Everything was then double or a confused mass, and he could only see well when closing one eye. There was no colour-blindness, and no trace of optic atrophy.

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[1] "British Medical Journal," Sept. 22, 1883.

Although in the majority of cases the symptoms are symmetrical, yet, in some cases ataxy as well as other symptoms, are confined to one side of the body in the second stage of tabes; and this condition has been called *hemi-ataxy*. Thus some patients have amblyopia on one eye, and say that they have a good leg and a bad leg. Pain and anæsthesia are sometimes confined to one side. In a case which was sent to me by Dr. Pearce, of Leicester, in July, 1880, the patient had had iritis in the left eye, had numbness in the left ulnar nerve, much more numbness in the left than in the right leg, tightness in the left side of the head, neck, and shoulder, and pain generally confined to the left side. He would, for instance, get a shoot of pain in the left wrist, which would, when at dinner, make him suddenly drop his fork ; but this did not occur in the right wrist. This peculiarity, however, generally becomes less marked as time goes on, when the other side becomes similarly affected. In a few cases, however, it appears to remain so throughout the whole course of the disease.

Several theories have up to the present time been brought forward for explaining physiologically the phenomena of ataxy which have just been described. Some observers, and more particularly Friedreich[1] and Erb[2] state that ataxy is a true motor symptom, and not in any way connected with, or dependent upon, the anæsthesia which is generally present at the same time. They base their views on the fact that in many cases the degree of ataxy is not at all proportionate to the degree of anæsthesia which may be found, inasmuch as the one may be slight, and the other severe, and *vice versâ*. A further apparent support has lately been given to this view by the discovery that neuritis of the peripheral nerves has, at least in some cases, to be looked upon as the cause of any high degree of anæsthesia which may be present (p. 30). Moreover, complete anæsthesia of one side of the body has been seen in hysterical women, and after certain cerebral lesions, without being apparently accompanied with ataxy. Finally there is a wonderful case, known as Späth-Schüppel's case[3] which will no doubt do duty for many years to come, like that of the Count de Lordat in another chapter of spinal pathology, and which is now by several pathologists believed to have settled this question for ever. The patient who was the subject of this remarkable case suffered from complete cutaneous, muscular, and articular anæsthesia, owing to spinal disease, while there was no trace of ataxy in walking. After death hydromyelus was found, and wasting of the posterior columns in the lower portion of the cervical cord, while the dorsal portion was only slightly atrophied, and the lumbar portion normal. The lateral columns, the grey commissure and the posterior horns were likewise much affected, and the posterior roots of the third to the eighth cervical nerves sclerosed, while the anterior columns, horns, and roots were normal. Erb has argued that if a normal condition of sensibility were really essential for co-ordination, there ought to have been a high degree of ataxy in this case, since anæsthesia was complete; there was, however, no ataxy, and he therefore thinks that sensibility is not indispensable for co-ordination; it may be necessary for first learning to co-ordinate movements, and is undoubtedly of importance for equilibration, but is unnecessary for carrying out complex movements which have already been learnt by training. He therefore assumes the existence in the cord of special co-ordinating centrifugal fibres, and thinks that ataxy is only observed when these centrifugal fibres are diseased. He appears to suppose that they are situated either in the central grey matter or in the lateral columns, but leaves this point open for future investigations.

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[1] Virchow's "Archiv," vol. lxviii., 1876, and vol. lxx., 1877.

[2] "Krankheiten des Rückenmarks," p. 85. Leipzig, 1876.

[3] "Beiträge zur Lehre von der Tabes Dorsualis," Tübingen, 1864, and "Archiv für Heilkunde," vol. xv., p. 45, 1874.

On the other hand, Leyden[1] and Pierret[2] have argued that tabes is really a disease of the sensory tracts only, and that all symptoms of motor disturbance which occur in it are to be explained by the influence which sensation is known to have on motion, or by the intimate relations existing between the motor and sensory tracts. Leyden finds the principal bases of this theory to be the following :—

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[1] "Ueber die graue Degeneration," etc., Berlin, 1863; and article "Tabes Dorsalis," in Enlenburg's "Encyclopädie," p. 57. Vienna, 1883.

[2] "Transactions of the International Medical Congress of London," 1881. Vol. ii., p. 399.

1. The portions of the cord which suffer in tabes are notoriously concerned with sensibility. This is more certain of the posterior roots than of the posterior columns. Van Deen has asserted that the latter are sensitive, but Schiff has proved the contrary. Their relation to the posterior roots, however, is undoubted from anatomical and evolutional considerations, Burdach's columns being the direct continuation of the posterior roots, while Goll's columns are generally considered to be the centripetal continuation of these fibres towards the brain. The principal bulk of the posterior columns is therefore composed of afferent fibres ; there are no positive reasons for assuming the existence in them of other fibres of unknown function, while the occurrence of centrifugal fibres in these parts is incompatible with the structure of the posterior columns.

2. Sensibility always suffers in tabes ; lightning pains, paræsthesia, and anæsthesia are constant symptoms.

3. A normal condition of sensibility is indispensable for normal co-ordination of movements ; and where sensibility is affected, co-ordination suffers likewise.

4. The affection of sensibility in tabes is, if not absolutely, at least tolerably proportionate to the degree of ataxy which may be present.

5. Certain nearly constant signs of tabes, such as Romberg's symptom, can only be explained by the part which sensibility plays in co-ordination.

Leyden disposes summarily of the objections which have been made against the sensory theory of tabes. When it is stated that there are cases of tabes with ataxy where even the most careful exploration of the different kinds of sensibility shows the latter to be unaffected, he replies that either the examination has not been sufficiently exact, or the cases have not been those of tabes. There are certain forms of acute ataxy and of Friedreich's disease which cannot be looked upon as appertaining to tabes proper. Again, it has been stated that an affection of motility which may be experimentally produced by interfering with sensory paths does not resemble typical ataxy; but this Leyden thinks of little consequence, the principal thing being that motility suffers when sensibility is impaired. Artificially produced conditions are in any case so different from tabes as a disease, that complete analogy cannot be expected. Finally, with regard to certain forms of anæsthesia which exist without ataxy, viz., those which occur in hysterical women and cerebral hemi-anæsthesia, he considers that hemi-anæsthesia proves nothing, and anæsthesia in hysterical women as good as nothing; while Späth-Schüppel's case appears to him to prove even less. A single case, with uncommon clinical features, and showing uncommon post-mortem changes, is as little conclusive as a single experiment which cannot be repeated with the same results.

It thus appears that the two principal representatives of German pathology of the present day differ *toto cœlo* in their views of this important subject. A careful critical consideration of all the different points in dispute seems therefore necessary in order to enable us to arrive at a satisfactory conclusion; and I will begin this difficult part of my subject with an examination of the data which are furnished to us by anatomy and physiology concerning the question now under discussion.

The anatomical and evolutional facts which have been given in the first chapter (pp. 8-10) appear very clear as far as they go. We have seen that the posterior columns consist of two different systems, one of which, viz., Burdach's columns, have to be looked upon as short conducting paths, while the other, viz., Goll's columns, are long conducting paths. Burdach's columns are direct continuations of the posterior root-fibres; they connect the cord with peripheral parts, and thereby with external influences. They also send out numerous fibres, which proceed in various directions into the central grey matter, and which are evidently intended to connect the different segments of the grey matter with each other, while others again proceed upwards into the medulla oblongata, where they terminate. Goll's columns, on the other hand, which are immediately contiguous to Burdach's columns, are long conducting paths, which proceed from the central grey matter of the cord up to the medulla oblongata, and appear from their anatomical peculiarities intended to connect extra-medullary centres in the brain and cerebellum with physiologically identical fibre-systems at different levels of the cord. It seems, *primâ facie,* reasonable to assume that the various commissures which are intended to establish a connection between extra- and intra-medullary centres, put all of these into mutual functional relations, and form a path for producing a physiological consensus between them. Destruction of these anatomical commissures by disease would therefore naturally be expected to lead to a cessation of at least some degree of functional harmony.

Experimental physiology, on the other hand, has given hitherto somewhat ambiguous results, which seem at first sight in contradiction to the data of normal and morbid anatomy. Schiff has shown that division of the posterior columns in animals causes loss of the sense of touch in all parts behind the lesion, but not loss of the sensation of pain, or analgesia. On the other hand, Goltz, who has studied the question of the localisation of the faculty of co-ordination, has come to the conclusion that no centres for the co-ordination of complex movements exist, either in the posterior columns, or in fact anywhere throughout the entire extent of the cord, but that such centres are to be found in the brain, more particularly in the corpora quadrigemina, the optic thalamus, and the cerebellum. As therefore the posterior columns do not appear to be the centres of co-ordination, we might at least expect them to contain the paths by which the co-ordinating impulses travel from the brain to the muscles. But even this supposition is controverted by Woroschiloff's experiments, which tend to show that, at least in the rabbit, these paths are situated in the middle third of the lateral columns of the cord, and do not touch the posterior columns at all.

The physiology of the *posterior roots* is somewhat better known than that of the posterior columns. If the posterior roots for one hind-leg are divided in a frog, the movements of that leg are seen to be out of harmony with the other, whether for jumping, swimming, or other modes of locomotion; it appears clumsy and inert; and when such an animal is held between the fingers, the affected leg will not make the movements intended for the purpose of escaping, as is done by the unaffected leg. After experimental division of the whole of the posterior roots, a frog, when put into water, is seen to be unable to swim; and, if incited to do so, gives himself up to ataxic movements without attaining its purpose. Locomotion is therefore seen to be deeply influenced by sensorial impressions imparting to the animal information about the position of different parts of its body; and when this information is uncertain, incorrect, or altogether absent, want of coordination is seen to follow.

It is important to notice that impressions which are simply and solely furnished by the skin are not indispensable for locomotion. Thus Claude Bernard has shown that a frog, whose skin was entirely removed, could still swim quite well; while destruction of the posterior roots at once put a stop to the power of swimming. This fact has a distinct bearing on those cases where ataxy has been observed together with normal sensibility in the skin. Our notions of the position of our limbs are evidently not only determined by the sensibility of the skin, but also of the more deeply situated structures, such as muscles, ligaments, cartilages, and bones, which may lose their sensibility independently of that of the skin. It is true that in many cases of hysterical hemi-anæsthesia the movements of prehension, locomotion, etc., are reported to have been normal; but there are other cases in which unquestionably ataxy of movements exists under such circumstances. In a girl, aged eleven, who is at present (June, 1884) under my care at the hospital, there was, on admission, hemi-anæsthesia of the entire left side of the body, which had apparently come on through hæmorrhage into the most posterior portion of the internal capsule at the time of birth. The affection had continued unaltered all her life until she came under my care, but yielded nevertheless to a single application of electricity. There had been no sign of paralysis at any time, and the girl was, on examination, found to be well able to carry out every simple movement with the anæsthetic limbs which I asked her to perform. At the same time *there was decided ataxy in the left hand.* The girl was able to play the piano with the right hand, but could not do so with the left; she had great difficulty in picking up a pin with her left hand, or do any useful complex action; yet the muscular force, as tested with the dynamometer, was quite normal. The use of the hand for finer movements improved only gradually after sensation had been re-established.

Vierordt and Heyd have shown that when the soles of the feet of a healthy person are rendered anæsthetic by the prolonged application of a freezing mixture of ice and salt, or by Richardson's ether spray, ataxy of movements is produced. This shows that, when no information is given by the nerves of the feet to the centres of co-ordination, these are unable to act.

Let us now analyse very briefly the mechanism by which walking is rendered possible under ordinary circumstances, and then proceed to consider the mode of production of ataxy in the lower extremities, where it almost invariably occurs first. Walking in a proper manner requires, in the first instance, the integrity of the entire motor zone of the brain and spinal cord. The great automatic centre of motor power and muscular nutrition and tonicity resides in the large ganglionic cells of the grey anterior horns of the spinal cord, some of which are of sufficient size to be visible to the naked eye when coloured with carmine. In these cells the power of motion is produced, and they send the force which constantly originates in them to the motor nerves and muscles by means of Deiters's processes or axis-cylinders. We have, therefore, at our command an instrument of motor power always ready to act, if called upon to do so. The volitional impulses for this originate in the hemispheres of the brain, and more particularly in the central grey convolutions bordering the fissure of Rolando. These automatic and volitional centres of motion are intimately connected with one another by a commissure of white conducting fibres, known as the pyramidal strands, by means of which the will acts on the medullary centres, and which communicate freely all the way down with the motor cells in the anterior horns. It is, however, not in the medullary centres that the action of the motor nerves and muscles is co-ordinated and regulated. This, on the contrary, is done in the central ganglia of the brain, viz., the corpus striatum and thalamus opticus, which communicate through the white internal capsule with the higher motor centres above and the lower motor centres further down. Of these the corpus striatum co-ordinates motor power, while the optic thalamus co-ordinates sensory impressions, and the internal capsule serves as a conductor. Both central ganglia acting in unison have the special function of rendering movements which are intimately connected with sensations, and which are in the first instance only excited by conscious volitional efforts, gradually mechanical and automatic. The object of this contrivance is to save time and trouble to the highest portion of the brain, or the grey surface of the hemispheres, which is intended to be habitually occupied only with the most important manifestations of life. Walking and all other complex movements have to be learnt early in life by countless conscious efforts on the part of the hemispheres; and full attention is necessary in the beginning to enable us to carry out such movements in a proper manner. But the older we grow, the more frequently we have directed our minds to all such forms of action, the less effort will eventually be necessary on the part of consciousness and volition; and ultimately all such movements will be performed mechanically, and without much, if any, attention to them on the part of the grey surface of the brain. A man, who is in the habit of writing much, never thinks of the way in which he forms his letters on the paper, over which his pen seems to fly quite mechanically. The same holds good for the various kinds of needlework, embroidery, playing the piano, the violin, dancing, riding on horseback, singing, decent eating and drinking, etc. If, each time we do anything of that sort, a conscious effort were necessary for all the different parts of which the action is composed, the time at our disposal would not suffice for a hundredth part of the work which we actually get through in life; and some forms of activity, such as finished piano and violin playing, would be utterly impossible. The act of walking, indeed, becomes in course of time so automatic that in general no attention whatever is paid to it.

In order, however, that the central ganglia shall be able to thus minimise the work which has to be done in life, it is necessary that they should constantly receive accurate information of the position of our limbs, and the nature of the obstacles with which the latter come in contact. We may be able to walk fast enough on a smooth level road without thinking about it, but if the pavement has been taken up, or we have to walk across a newly ploughed field, or on the edge of a precipice, or on a narrow bridge or plank thrown across a stream, or in a crowded thoroughfare where hansom-cabs, omnibuses, perambulators, tricycles and foot-passengers jostle each other, or in the dark on a staircase with which we are not acquainted, then a considerable amount of attention is required for overcoming such obstacles in our way with safety. The mere impressions conveyed to the central ganglia by the posterior columns are then no longer sufficient, but the aid of the eyes, or, in the case of the dark staircase, of the hands and arms, is instinctively called in, in order to supplement the ordinary sensory impressions by special information and subsequent manœuvring. We, therefore, under those circumstances behave like the ataxic does habitually, that is to say, we use our eyes as crutches, and manœuvre with our hands and arms to assist us ; and even then we do not walk as well as we do on a smooth, level road where there are no impedimenta of any kind to be overcome. The ataxic, therefore, is habitually in the condition in which we are under such special circumstances as I have just mentioned. The information habitually given to the central ganglia by the posterior columns is not available for him, because those columns have ceased to exist, and the various groups of ganglionic cells can therefore no longer be combined for synergic, orderly, or purposive action. There is no longer any harmony between the muscles which act and their antagonists which regulate the action ; wrong groups of muscles are called into play, which impair the action instead of facilitating it ; the antagonists act too energetically, and those muscles which produce the action have therefore to redouble their efforts in order to arrive at a certain result. There is, therefore, useless expenditure of nervous force, causing fatigue, which latter is increased by calling in the aid of the central convolutions bordering the fissure of Rolando. In order to be able to walk at all, the ataxic has to use his eyes as crutches, and his arms and hands as manœuvring-poles; and even then, when he is spending all the reserve forces stored up in the nervous system, he will court failure, and stammer with his feet.

The mode of production of locomotor ataxy thus appears satisfactorily explained; and it only remains for us to account for the phenomena of static ataxy which are generally associated with the former.

The cerebellum, which was once believed to be the seat of the reproductive faculty and desire, is now known to be the centre of equilibration of the body. Removal of the cerebellum in an animal causes static ataxy; the animal cannot keep steady on its legs, but staggers about as if it were drunk. It is not paralysed, and endeavours to carry out certain movements, but there is an utter want of precision, and even the most desperate efforts do not succeed in steadying it. We have already mentioned (p. 182) that one portion of this organ prevents us from falling forwards, another from falling sideways, and from constantly turning round in a circle, while a third is intended to secure us from falling backwards. The behaviour of animals deprived of their cerebellum in fact resembles in the closest possible manner that which we have seen to occur in ataxy. The erroneous information which the cerebellum receives from the diseased cord may, however, be to some extent corrected by sight; and this accounts for the fact of standing being so much more difficult when the eyes are closed (Romberg's symptom), as well as for the other phenomena of static ataxy. Whether the paths through which information is given to the cerebellum are situated in Goll's columns, in which case the road would be somewhat more indirect, or in the direct cerebellar strands, which would carry information in a straight line to the cerebellum, we are at present not in a position to determine. We may, however, take it as an indisputable fact *that the symptom of locomotor ataxy is caused by an interruption of the paths between the posterior roots and the central ganglia of the brain through* *sclerosis of the posterior columns, and that static ataxy is in its turn brought about by an interruption of the paths between the posterior roots and the cerebellum, through sclerosis either of Goll's columns or of the direct cerebellar strands.*

23. *Sensibility in the second stage of tabes.*—a. The *lightning-pains* have been already so fully described (p. 146) that it is not necessary to say anything more about them, except that they may continue throughout the entire second period of tabes. In some cases they appear to increase in intensity and frequency of occurrence as time goes on; while in others gradually longer intervals of rest are noticed. As a rule they diminish or disappear altogether when the second period begins to merge into the third, and when it is to be supposed that most or all the fibres which are concerned in the production of these pains have been destroyed. Sometimes these pains leave the lower limbs and attack the arms, as time goes on; which is to be accounted for by the tendency of the disease to spread upwards in the spinal cord. When all the fibres in the lower portion of the cord which transmit the sensation of pain have been destroyed, anæsthesia and analgesia take the place of lightning-pains; but at this period there may be active irritation still going on in the upper portion of the cord, causing lightning-pains in the upper extremities, so that the patient is no better off.

b. *Numbness*, which is generally slight in the first stage, is habitually more pronounced in the second. The patient complains chiefly of numbness in the feet; and when asked to describe the sensation more minutely, says that there is a kind of heaviness or furriness, as if they had gone asleep; or as if the feet were in thick fur boots, or rested on thick woolly rugs or carpets or water-cushions. The contact of slippers, boots, and drawers is not properly felt. The degree of this numbness varies from time to time, is worse when a change in the weather is impending or during storms, or when the patient is fatigued: it is apt to spread as the disease progresses, and gradually invades the ankles, the legs, thighs, hips, and goes up to the waist, where it encounters the peculiar form of tightness which has already been described (p. 201). In the upper extremities, on the contrary, the numbness is habitually perceived in the third and little finger, and is apt to spread from there on the ulnar side of the forearm up to the olecranon, beyond which it often does not pass for many years. It also affects the entire palm of the hand, while numbness in the lower limbs is generally equally pronounced in both sides; it often remains confined to one of the upper extremities in the region just mentioned, and sometimes invades the other gradually in a different part, so that the thumb and first finger are particularly affected. It generally precedes the symptom of ataxy by a short interval, and is proportionate in extent to the degree of ataxy which may be present.

Numbness is one of the most constant symptoms of the second period of tabes, and is only very exceptionally absent. It is important to know that it is frequently present without any form of anæsthesia which may be objectively determined. Indeed, it is a symptom *per se*, concerning which we have to rely entirely on the statement of the patient.

c. *Anæsthesia*, or loss of the sense of touch or contact, is a frequent symptom at this stage of the disease. We can determine this objectively by examination of the patient; but we should not rely too implicitly upon his statements unless he happen to be a particularly intelligent person. In people of ordinary intellect a second examination gives occasionally entirely different results from those of the first; and it must be confessèd that no examination takes more time, is more tedious to both patient and doctor, and, even when systematically performed, eventually more uncertain in its results than that for anæsthesia and its various degrees.

It is important that a definite method should be followed in testing a patient for anæsthesia, as the rough-and-ready mode which is often thought all that is required, viz., to prick the patient with a pin, is by no means sufficient. When we wish to discover slight degrees of anæsthesia, we request the patient to close his eyes, and then very gradually approach our first finger to that part of his skin which we intend to examine. We satisfy ourselves first that there is no considerable difference of temperature between the examiner and the one to be examined; our own hand must never be colder than the patient's skin, as the sensation of cold may still be keenly felt when the perception of ordinary contact is lost. As soon as we ourselves perceive the least sensation of contact, the patient should do the same. We must avoid rubbing or pressing the skin, or even a sudden approach, as all such manœuvres imply more than simple touch. After satisfying ourselves whether the patient feels our touch or not, we next ascertain whether he feels a difference between wet and dry, smooth and rough; and, in the hand and fingers, whether the patient can tell the nature and shape of certain familiar objects, such as coins, knives, pens, pins, etc.; we also examine the patient with Weber's compasses, or æsthesiometer, remembering the normal distances for the perception of the two separate points of the instrument, which vary from half a line on the tip of the tongue, and one line on the volar surface of the third phalanx of the fingers, to two inches and a half in the arm and thigh. In order to test the common sensibility of the feet, we let the patient stand on a soft rug and a bare board alternately, likewise with his eyes closed, and ascertain whether he feels any difference in them.

Loss of tactile sensibility occurs generally some time before the sensibility to pain or to cold is diminished. It is chiefly found in the soles of the feet and the palms of the hands, and seems proportionate in degree to the distance at which the parts are from the posterior roots. It is therefore more marked in the foot than in the leg; more in the leg than in the thigh; more in the fingers and hand than in the arm and shoulder, etc.

Anæsthesia may also be present in the entire sphere of the fifth nerve. It is occasionally confined to one side of the body, and may appear in irregular patches. The patient is unable to take any special object out of his pockets, if there are several different things in them; he may bring out a pencil-case or knife when he wants a shilling, etc. The sense of touch may also be perverted, so that simple contact feels like pain, or sends a shiver through the patient, who may continue to feel it for several minutes after the touch has been made.

Where a tabid patient has become totally blind and stone-deaf, he may still entertain some communication with the outer world by means of the sense of touch. Strümpell has seen a case where only by touching the forehead the man could be made to understand anything. One letter after another was traced on the forehead with the finger; and the patient had gradually gained considerable facility in making out such words. He at once pronounced aloud the letter which had been traced; if it was correct, another letter was traced, but, if incorrect, the hand was drawn across the forehead, and the letter repeated until he made it out. If a word or sentence was finished, a simple tap on the forehead informed him of it. A tap also meant "yes," while drawing the hand across meant "no" or "wrong." It was thus possible, with time and patience, to have some conversation with this man. As he was an intelligent fellow, he guessed most words after one or two letters had been traced; for instance, when at the visit of the physician "d-o-" had been traced, he at once said, "I'm sure it is the doctor! Good morning, doctor; how do you do?" Curiously enough he guessed the letters traced by the nurse's hand much better than those by the doctor, probably from the former corresponding more with his own previous way of writing. He often inquired after the weather, the time of day, etc. ; otherwise he lay quietly in bed, with his hands covering the face, apparently in order to gain a certain amount of consciousness of his body.

Schiff has shown that tactile impressions are transmitted to the brain exclusively by the posterior columns of the cord. Division of these columns in rabbits, in front of that part which gives rise to the nerves for the hind legs, prevents any tactile impressions made on those legs from being perceived by the animals ; while, on the other hand, division of the whole cord, with the only exception of the posterior columns, leaves tactile sensibility unimpaired.

d. *Analgesia, or loss of the sensation of pain,* is likewise common at this period. Sensibility to pain is examined by pricking the patient with a pin, pinching him, and by applying the different forms of electricity. With regard to the latter, both the induced and constant current may be employed. The best mode of using faradisation is to apply a perfectly soft faradic wire brush with a large surface (such as made for me by Coxeter and Son) to the point to be examined, while another moistened electrode is placed on the sternum or the nape of the neck. The minimal current-strength at our disposal is then applied, and this is gradually increased until a decided, although slight, sensation of pricking and heat is noticed. The distance of one coil from the other is then read off at the stem provided for this purpose, and compared with the other side of the body of the patient and healthy averages. The continuous current has to be applied by two moistened conductors, the large anode being placed on the sternum or the nape of the neck, and the smaller cathode on the point to be examined. As soon as a feeling of heat and pricking is experienced under the cathode, the number of milliampères, or fractions of such, is read off at the galvanometer, and compared with the corresponding part of the other side, and with healthy averages. This mode of examination yields the most certain results; but it takes up a good deal of time, and cannot be carried out by any but specially skilled observers.

Loss of the sensibility to pain is generally a later symptom than anæsthesia, probably because tactile impressions are not so keen as those giving actual pain; so that the latter may still be perceived at a period when the simple sense of touch appears to have already vanished. Cruveilhier mentions the case of such a patient who fractured his leg, and neither at the time of the accident nor afterwards felt any pain whatever.

Analgesia is frequently incomplete. Thus, the prick of a pin may be felt as a simple touch, while the patient does not feel the head of the pin at all. If the excitant used is very powerful, pain may still be felt when ordinary painful impressions are no longer resented. This holds good more particularly for the proceeding known as faradisation of the skin, which, when a powerful current is used, is almost invariably felt, even in the third period of the disease. It may even then, if continued for some time, rouse any feeble remnant of sensibility which may be left, so that sensibility to ordinary touch, or the prick of a pin, returns for a time. It seems as if the few remaining healthy nerve-tubes, which may still exist at different levels of the posterior roots or columns, were by such a proceeding shaken up from their habitual torpor. Sensibility, thus restored, however, generally disappears again a few hours afterwards, and a dead level of analgesia is re-established; nor does it, at the time when faradisation is used, come back at any other place, except in the one which has been directly touched by the brush. This is a point of difference between tabid and hysterical anæsthesia, as in the latter we may, by faradisation of the skin of a limited area of the forearm, restore all forms of sensation in an entire side of the body, and that more or less permanently.

About this time we may also observe the peculiar symptom of *anæsthesia dolorosa.* This sign, which appears *primâ facie* unintelligible, is owing to two different conditions, viz., first a considerable diminution of sensibility, and simultaneous hyperæsthesia produced by unusually powerful stimulants. If, for instance, a portion of the skin which is no longer sensitive to touch is strongly pinched, this pinch will not only be felt, but will even be felt more unpleasantly than if the skin were in its normal condition; while at the same time a special feeling of burning is experienced, which is quite unbearable to the patient. A similar state may be artificially induced by making a leg "go to sleep," through squeezing the sciatic nerve between the sciatic notch and the edge of a chair. We have then anæsthesia of the skin; but if the latter be pinched, there is, in addition to the pins and needles, a peculiarly unpleasant burning sensation, showing that the sentient nerves have undergone a peculiar modification.

The curious phenomenon of *delayed sensation* is chiefly observed where there is analgesia, and less where there is common anæsthesia. The impression is then experienced from one to five, seven, and even ten seconds after it was made. This delay in perception is greater in proportion to the distance of the part acted upon from the posterior roots.

How can we physiologically explain this curious symptom? As the rate of transmission of nervous power in man is about forty yards per second, an impression made upon any part of the body, however distant from the centre, is practically perceived at the same instant that it is made. But in tabes there are impediments to transmission. Unless there were peripheral neuritis at the same time, we would expect the impression to travel up, with its normal rapidity, as far as the spinal ganglia; but in the posterior roots an obstacle is encountered, which is likewise present in what remains of the posterior columns. It is probable that, although the central grey matter of the cord is, according to Schiff, æsthesodic, yet the transmission of impressions to the brain would in general rather pass through the posterior columns than the central grey matter, for the sake of increased speed. In the central grey matter the road is more encumbered, as there the impression must travel from one ganglionic cell to another by means of Deiters's prolongations, which connect one cell with another, while the transmission along the course of straight nerve-tubes in the posterior columns must be infinitely more rapid. In tabes, however, many, or most of the fibres of the posterior columns have been destroyed, and are replaced by connective tissue which does not conduct at all. We have, therefore, a similar condition as if, in a galvanic battery, the conducting copper wires had been replaced by ordinary twine, which is no conductor. The current would, therefore, seek another way, although it might be much more roundabout than the direct road which has been blocked up. In the cord this road passes through the central grey matter; and it may be easily imagined that if there should be likewise impediments in the way there, the transmission would then reach its extreme limit of delay. We have seen (p. 22) that the posterior cornua, and the place of junction between the anterior and posterior cornua and Clarke's vesicular columns, are frequently found affected in tabes, and these are just the portions of the central grey matter which are more particularly concerned with sensation. Should they be entirely destroyed, in addition to the posterior columns, it stands to reason that any transmission of sensitive impressions to the brain is rendered impossible.

e. *Loss of the Sense of Temperature.*—This sense is best examined by applying test-tubes filled with hot, tepid, and cold water, to the skin. A test recommended by Erb is to blow on the patient's skin from different distances; blowing near is felt warm, and blowing from afar gives an impression of cold. In cold weather the impression of any metallic objects which are at hand may be utilised. Ice and Richardson's ether spray may also give useful indications. Eulenburg has constructed a special instrument for conducting such investigations, which he calls the *thermæsthesiometer*, which may, however, in general be dispensed with.

Persons in good health are able to distinguish with certainty a difference of one, or even half, a degree. Objects which are warmer than 93° give a sensation of heat, and such as are below 90° feel cold. This faculty of distinguishing different temperatures remains occasionally unimpaired in the tabid, although they may have lost all other kinds of sensations. Topinard relates the case of a patient who was affected by double amaurosis, absolute anæsthesia and analgesia, with ataxy of a severe degree, and who had only the sensation of heat and cold left to tell him that he still had his limbs. Sometimes his legs would be jerked out of bed by spasms which he did not feel, and which, being blind, he could not see. Then, after a time, a sensation of cold would creep upon him, and the poor fellow would ask whether anything was the matter with his legs. This patient lived only by his memory, as he had lost the consciousness of his body. But the sense of temperature may also be wanting. Leyden mentions the case of a patient who prepared a warm bath for himself, and not being able to distinguish between heat and cold, made it very hot, and was severely scalded on going into it.

It is a singular circumstance that the sensibility to *cold* persists longer in tabes than almost any other form of sensibility. Indeed, it is frequently found exaggerated at a time when the sense of touch is entirely lost, and when there is already a considerable degree of analgesia. Thus the contact of a piece of metal or of a sponge saturated with cold water may be perceived, when neither ordinary touch, nor the prick of a pin, nor a gentle electric current are felt any longer. The impression of cold is indeed often so sudden and surprising to the patient that extensive reflex movements are caused in consequence. It is difficult to account for this curious symptom. Vulpian thinks that it may be owing either to an undue degree of excitability of the grey matter of the cord, where such sensations are elaborated, or to exaggerated vibrations produced in the cutaneous nerves, in consequence of traversing posterior root-fibres which are in a state of irritation. This, however, is really no explanation, but only a different way of stating the fact that the excitability to cold is exaggerated; for why should not the irritation of the central grey matter or the posterior roots give rise to exaggeration of the other forms of sensibility? Other observers have thought that there are special sets of nerve-fibres for perceiving temperature, and others for pain and contact. This is also most unlikely; for if there were any such, why should they escape the morbid influence? Another theory is that there are certain extra-medullary centres which serve for the reception and elaboration of thermic impressions, and that these are not invaded by the sclerosis. This is equally unsatisfactory, for we may well ask why should they be thus exempt; and should also be obliged to assume the existence of special intra-medullary centres for the perception of heat as well as cold. This at once reduces the proposition to an absurdity. We must, therefore, for the present be satisfied with knowing that cold is one of the few stimulants the response to which, in tabes, is generally increased, instead of diminished.

It should also be noted that the transmission of the impression of cold is less delayed than that of touch or pain. In advanced cases, however, it is likewise delayed, and more especially so if the impression be made nearer to the periphery than to the centre.

f. The sense of *locality* may be examined by touching or pricking the patient in a certain spot, his eyes being at the same time closed, and then letting him indicate the point touched or pricked with his finger. This modification of sensibility is likewise frequently found to be diminished in the second stage of tabes. Diseased structures conduct impressions made on the skin, not in the definite mode in which this is done by healthy structures, but in an uncertain and halting kind of manner which does not allow of a clear perception of the locality of the point which may have been touched. It is a curious circumstance that if the patient makes a mistake in localising an impression, he almost invariably points to a place nearer the centre than the one which has been touched. He is sometimes wrong to the extent of several inches.

g. The sense of *pressure* may be investigated by putting different weights on various parts of the patient's body, when he will occasionally be unable to tell the difference between an ounce and a two-ounce weight. Eulenburg has constructed a special instrument for investigating this peculiarity, which he has called the *baræsthesiometer*. This shows differences in pressure by the movements of an index on a dial. The ordinary post-office scales and weights will however, generally be found sufficient. Coins or billiard balls of different weights are also useful. This sense is equally affected in tabes as the other modes of sensibility.

h. *Tickling* the soles of the feet, the knees and other parts is occasionally not perceived at all, while in other patients a curious thrill and shiver is sent through the whole body, the impression of which may last for a considerable time.

The singular manner in which these different forms or kinds of sensibility are affected in tabes has led some pathologists to the conclusion that there are different nerves for each one of them—some for contact, others for pain, others for temperature, tickling and so on ; and Brown-Séquard has distinguished the alarming number of twenty-two different kinds of central nerve-fibres. There is, however, no evidence whatever to show that all these different or special nerve-fibres with highly specialized physiological attributes exist; on the contrary, it appears certain that the same conductors may transmit to the brain all the various kinds of impressions which may be made on the peripheral expansions of the sentient nerves.

Vulpian has shown that not every one of the individual sentient nerve-tubes ascends to the cord through the spinal ganglia, since the bulk of the posterior root is not the same throughout its extent. If there were a real continuity of fibres all the way up, then we should no doubt see definite and absolute anæsthesia involving all the different kinds of sensibility in certain well-defined areas, while in other equally well defined areas all forms of sensation would continue. Such, however, is not the case. The connexion between the peripheral fibres and the posterior root-fibres is therefore only indirect, so that every root-fibre may be caused to vibrate by impressions made on any peripheral fibre. When there has been destruction of root-fibres through sclerosis, the vibration which any peripheral fibre may convey to the cord and brain cannot be so keen as it must be when all root-fibres are in full physiological activity. If, therefore, the impression which is made be slight, it will not be perceived, while a more vigorous impression may still be felt. We are, therefore, led to the opinion that the peripheral afferent fibres, as well as the posterior roots and nerve-cells in the spinal ganglia, as well as the posterior grey matter of the cord, have the same physiological properties; and that the differences observed are more in the nature of the excitants or stimulants used, than in the different portions of nerve-structures themselves. Different stimulants cause different molecular vibrations in the nerve-tubes as well as in the grey cells; and this is the reason why contact, painful impressions, heat, cold, etc., are differently appreciated.

i. *Muscular sensibility* is likewise affected at this stage. Squeezing the muscles and faradising them by a weak or moderate current, with the aid of moistened conductors, is often not perceived. The impairment of this sense is no doubt intimately connected with another curious symptom which is often observed, viz., that the patient has entirely lost the faculty of knowing where his legs or arms are. He appears to lose them in bed; he cannot tell whether they are flexed or extended, crossed or lying side by side, or, if crossed, which is crossed over the other. He has actually to search for his legs with his fingers from the hips downwards, in order to be able to give an account of their position. It is, however, probable that the sense of the position of the limbs does not exclusively reside in the muscles, but also to some extent in the skin, the joints, and the bones.

24. *Other reflexes than the knee-jerk* may likewise be much diminished or lost. Cutaneous or superficial reflexes as a rule keep pace with the state of sensibility. Where the latter is much affected, these reflexes, more especially those elicited by tickling the soles and the knees, are apt to become sluggish. The cremasteric, abdominal, and epigastric reflexes are likewise often found to be lost in the later stages of the disease. The intra-ocular sympathetic reflex is generally absent,—that is to say, irritation of the skin of the neck does not enlarge the pupil.

The reflexes which continue unimpaired longer than any other are those which are produced by the influence of cold. If we touch, for instance, the inner surface of the thigh with a sponge full of cold water, or a cold metal, the limb is at once withdrawn, and sometimes this reflex takes place a few seconds before the sensation of cold is perceived. There may be quite a series of flexions and extensions of the legs and thighs, which resemble the movements of attempted escape in animals. Occasionally it is sufficient simply to uncover the patient when he is in bed, to make his legs move about, or, as Vulpian has aptly called it, gesticulate in all directions. If the sense of temperature is, however, entirely lost, these reflexes can no longer be elicited. All the other tendon reflexes disappear at a comparatively early stage of the malady.

25. The *urine* is in the beginning of the second period of tabes occasionally quite normal. In other cases there is an excess of phosphates and lithates; but what seems more important is the *presence of sugar*, which I have noticed in several cases. I have generally found this combined with the presence of an excess of urea, and the specific gravity of the urine has under these circumstances ranged between 1030 and 1037. The quantity of sugar in the urine appeared to vary considerably, amounting occasionally to nearly 200 grains in the pint. I have also known patients to discharge sugar habitually for two or three years, and then to cease doing so, showing that it is glycosuria rather than diabetes.

In other cases the urine contains myco-pus and leucocytes, and is liable to speedy ammoniacal fermentation; but these graver symptoms occur more especially in the terminal stage of the disease.

* * * * *

III. *Symptoms of the Third or Terminal Stage of Tabes Spinalis.*—In this stage the ataxy of movement is replaced by paresis or paralysis; and there is a proportionate increase in the severity of all the other symptoms, with the only exception of lightning and other pains, which as a rule vanish when all the fibres of the posterior columns have been destroyed. We observe then more or less complete paralysis of the bladder and bowels, absolute impotency and loss of sexual desire, a considerable degree of muscular atrophy and arthropathy, and tendency to bedsores. Brain-disease is now likewise apt to appear, and this requires special consideration.

26. *Late Brain-Troubles in Tabes.*—In some cases the intellect continues unclouded to the very last, while in

others it becomes affected in a variety of ways. It is not uncommon to find that patients, when they ultimately lose all hope of recovery, become extremely depressed in spirits. I have known such a patient to cry all day long like a child. In other cases there is melancholia; the patient becomes taciturn, shy, refuses to speak, or to take notice of anything; and this condition may alternate with fits of maniacal excitement. Where there is any hereditary predisposition to mental affections, this is more particularly likely to occur.

I will now shortly relate two cases of mental affection during the last stage of tabes, which were under my care some time ago:—

Case 70.—A gentleman, aged forty-five, single, consulted me in September, 1880. He had had syphilis in 1869, and obstinate secondary symptoms in the throat and skin. He told me that he had swallowed hundredweights of iodide of potassium and mercury, and had lately had a hundred inunctions at Aix-la-Chapelle, without the slightest benefit. Two years ago he had double vision from paralysis of the right rectus externus, and shooting pains in the lower extremities. Soon afterwards he lost the use of his legs, and the power over the bladder. He has still some amount of muscular force in the legs, but is utterly unable to walk or to stand. There is great numbness, anæsthesia and analgesia in the legs. The urine is habitually drawn off by the catheter, and there is such a degree of urethral anæsthesia that he does not feel the introduction of the instrument. The urine is ammoniacal, and contains a large quantity of ropy mucus. On coughing and sneezing there is always an involuntary spasmodic ejection of urine. The bowels are confined, and he does not feel the approach of any action, so that the evacuation generally takes place before he has time to reach the commode.

The patient improved for a time considerably under treatment, which consisted chiefly of the administration of nitrate of silver, ergot of rye, and electricity. The urine became at first feebly, and afterwards fairly, acid; the patient regained the power over the bladder to such an extent that he passed six or eight ounces of urine in a stream by himself, and retained it a great deal better. The action of the bowels was also much improved. He now felt a warning before a motion was going to take place, and got sufficient notice to avoid mishaps. The sensation in the legs likewise improved; he began to localise impressions, felt stronger generally, had a good appetite, and slept splendidly. He could walk tolerably well, when supported on both sides. At this time there occurred unfortunately a change in the attendance on the patient, with the result that he was induced to take sherry freely, beginning early in the morning, and going on throughout the day. This interfered at first with his sleep, and he became so restless, that he was in and out of bed all night. On November 20th, regular hallucinations commenced which were to a great extent of a terrifying nature. He screamed "murder" in a stentorian voice, so that it was heard at a considerable distance. He told me that his nurse had entered into a conspiracy with some other people to poison him; and that even his dog had been drugged. He, therefore, refused food of any description. I tasted some of his tea, which was standing by his bedside, and which he said was poisoned, in order to show him how groundless his fears were; and after that he drank some of it. The next day, however, he insisted on the nurse being discharged, saying: "The rascal told a lot of lies!" He then began to suspect myself, said that I was as bad as the rest, the medicines prescribed were poisons, and refused to answer any questions or to enter into any argument. I now retired from the case, and handed the patient over to a physician who had previously attended him. In the meantime his relations were telegraphed for, and on their arrival in town stopped the supply of liquors. In a few days the patient became rational again, and I was then requested to resume my attendance. When I saw him again, he frankly apologised for his conduct, and expressed his fears that I should never forgive him. On being reassured on this point, his mind became more easy, and he appeared to improve in various ways for some little time. On December 20th, however, he had several epileptiform fits, with total loss of consciousness, and great muscular rigidity. These fits increased upon him, his helplessness became greater from day to day, he wandered in his mind, and he died on December 30th, having for some time previously been in a comatose condition.

In the following instance symptoms of general paralysis of the insane became developed in the second stage of tabes :—

Case 71.—In March, 1882, Dr. Grasemann asked me to see a piano-tuner, aged 46, single, who had contracted syphilis in 1870, and had suffered severely from it. He had, however, apparently recovered from that disease, when in 1875 he began to complain of a feeling of constriction round the chest, which was thought to be connected with liver and stomach derangement. Some time afterwards lightning pains appeared in the legs, and ataxy of gait gradually became developed. The patient's mind was decidedly confused when I first examined him. There was no affection of the cranial nerves. Ataxy existed in both the upper and lower extremities. The legs were flabby and thin ; there were constant feelings of pins and needles, and, occasionally shooting pains in the limbs. The sexual power was lost ; the bladder in a state of atony, with occasional loss of control over the retention of the urine ; and constipation was habitual. The knee-jerk was absent in both sides. Shortly after he had consulted me, the patient was recommended by his friends to go to Aix-la-Chapelle to undergo the treatment for which that place is so well known. He had only been there for a few days, when severe symptoms of mental derangement manifested themselves, and it was found necessary to place the patient under restraint. I have since understood that the symptoms of general paralysis of the insane became developed shortly afterwards.

These two cases, to which many others could be added, show the statement current in the older treatises on tabes, viz., that the mental faculties remain undisturbed till the end, to be inconsistent with facts. This was first ascertained by French alienists, such as Baillarger, Magnan, Falret, and afterwards more especially studied by Rey[1] and Rougier;[2] and several forms of brain affections have since been distinguished.

There is a class of cases in whom simple impairment of the intellect is a prominent feature. The patient is no longer able to attend to his affairs; there are no delusions, but imbecility, which gradually becomes deepened: he has no will or desire of any kind, appears to be perfectly happy, and gives no trouble at all. His memory seems to be a blank, and if he still speaks, he is apt to stammer, and does not finish his sentence. The facial muscles appear in a state of tremor. Eventually coma supervenes, and after death meningo-encephalitis is discovered, together with the cord-lesion characteristic of tabes.

In other cases, the condition which has just been described is more temporary, and apt to come and go. There is then such a singular jumble of cerebral and spinal symptoms that the diagnosis may be rendered difficult, more especially at first, and if we are consulted at a time when the cerebral symptoms predominate. In these cases we find another illustration of the immense diagnostic importance of the loss of the knee-jerk. Whenever Westphal's symptom is found to be present, we may be sure that there is an affection of the posterior columns of the lumbar portion of the cord, whether ataxy exist or not. This latter point is sometimes difficult to decide, as in some forms of awkwardness of gait the symptoms may point as much to paresis as to ataxy.

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[1] "Annales Médico-Psychologiques," 5th series, vol. xv. Paris, Sept., 1875.

[2] "Essai sur la lypémanie et le délire de persécution chez les tabétiques." Paris, 1881.

Westphal finds that, out of a hundred patients who die of general paralysis of the insane, twenty have sclerosis of the posterior columns at the same time. Whether the symptom of ataxy is present in these, depends upon the intensity of the disease in the cord, and whether there is much affection of the posterior roots. Some patients die of the brain-disease before the affection of the cord has made much progress. Ataxy of gait only becomes developed when a large number of central nerve-tubes are dead.

Most patients who suffer from tabes, with temporary attacks of mental derangement, have a history of syphilis. Indeed we may say that the more multiple the symptoms, the more probable is the syphilitic taint in a patient, for syphilis has the tendency to produce multiple lesions in different areas of the nervous system. The characteristic feature of these cases, however is, that the *spinal symptoms are constant and permanent, while the cerebral symptoms are more or less temporary and fleeting.* The cause of this is obvious. Tabes is, even in the beginning, at once connected with structural lesions in the cord; while cerebral syphilis creates a tendency to occasional attacks of hyperæmia, or ischæmia, which cause alarming symptoms at the time being, but are apt to disappear spontaneously or under the influence of treatment.

Magnan has related a case in which the patient, aged forty-two, came from a healthy stock and had lived temperately, until in 1863 he contracted a hard chancre, which was quickly followed by roseola and condylomata about the mouth. This yielded to specific treatment, and he remained well until 1867, when he was suddenly seized with lightning-pains, to which were soon added other symptoms of tabes, viz., constriction of the chest, vesical crises, sluggishness of the bladder, plantar anæsthesia, Romberg's symptom, and incoordination in the lower, and after a time, in the upper extremities. In 1880 the intellect became impaired; the patient was hypochondriacal, and had delusions of persecution. He said that his body had lately begun to smell very badly, and his companions had therefore entered into a conspiracy to get rid of him; they walked at night in a procession round his bed, brandishing red-hot pokers; called him abusive names, put absinthe into his cocoa, &c. A similar case is recorded by Falret. It was that of a man, aged forty, with a history of syphilis, and who had strabismus of the left eye in 1872. Next year the right eye was similarly affected; in 1874 he had a stroke of hemiplegia. Lightning-pains and weakness in the lower extremities then supervened, and his mind became unhinged. In 1879 he was admitted into Bicêtre, with a number of symptoms of tabes, to which occasionally maniacal excitement, with grandiose delirium, was added. He imagined that he had inherited several millions of money, with which he intended to rebuild Paris; he was able to resuscitate the dead; was President of the Republic and King of Italy, had invented the perpetuum mobile, and a means of preventing the collision of steamboats. He was also subject to visual hallucinations; on one occasion he saw a chapel full of sisters of charity praying at the altar, with priests conducting the service; when suddenly all this disappeared, and an old woman came from the kitchen and blew in his face with a pair of bellows.

Rougier has given an interesting theory of the rationale of the development of some forms of mental disease which are observed in the later stages of tabes. According to him, they occur chiefly when so many portions of the nervous system are affected that morbid impressions perceived in one sphere can no longer be corrected by another. Thus a tabid patient when walking on deal boards, believes that he is walking on wadding ; but, provided his sight be good, he has only to look to his feet and see that he is really walking on boards. As soon, however, as blindness, deafness, and anæsthesia are added to his other symptoms, he may not only readily acquire erroneous impressions, but also be convinced of their reality.

The tabid leads a fearful life. He has often hardly words enough to describe the tortures which he constantly undergoes. He feels as if the flesh were torn from his bones, as if his inside was burnt with fire, as if he were being impaled, bitten by dogs and struck by daggers.[1] Apart from these painful sensations, however, he may, if the optic nerves begin to suffer, see stars, sparks, flies running, or blackbirds flying about, and fancy that he has dust, pieces of coal or other foreign bodies in his eyes. When deaf, he perceives tinkling of bells, rolling of drums, explosions of gunpowder and singing of birds. Taste and smell may be so affected that all kinds of food, even the most tasty and delicate, become nauseous and horrible ; and he commonly believes that fæces have been mixed with the food. As for touch, he often cannot distinguish between the bedclothes and the bedstead.

In consequence of there being hardly any but painful and unpleasant sensations, the delirium, where it occurs, is of a depressed character. The patient imagines himself persecuted ; the fearful pains from which he suffers, are attributed to imaginary enemies ; he says that he is tortured, poisoned, insulted, threatened, magnetised, choked by horrible smells ; that his life is being attempted ; that his feet are being cut off ; that rotten eggs, sulphur, phosphorus, mineral acids, vermin-killer, mud, fæces, arsenic, dynamite and verdigris are put into his food ; that frogs and guinea-pigs are placed into his bed ; that he is made to swallow boiling oil, which runs all the way through his inside and out again at the anus; that fæces are put into his legs; that his stomach is exploded by electricity, etc. The fog or mist, due to optic atrophy, presently becomes a cloud, then a blanket, then a ghost, and finally the ghost speaks to him. The idea of persecution will easily present itself to a mind unhinged by such varieties of strange sensations.

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[1] *Vide* description of "Lightning Pains," p. 146.

Sometimes there are regular attacks of delirium of this kind coinciding with attacks of hyperæsthesia, and the former disappear when the latter subside. There is, therefore, always a material base of suffering and distress in the sphere of the sentient or sensorial nerves; but the symptoms from which the patient suffers are erroneously interpreted by him. This condition is obviously quite different from general paralysis of the insane. There is no affection of language; the patient expresses himself with the greatest volubility, and has not the slightest difficulty in finding his words or finishing a sentence; there is no tremor in the facial muscles; the memory is good; and there are perfectly lucid intervals.

At this period of the disease, the patients become very vulnerable, and lose their power of resistance to morbid influences. The nervous system has now become incurably altered, and the trophic influence of the spinal cord is extremely reduced. They, therefore, easily die of intercurrent affections, such as bronchitis, typhoid fever, and sometimes of simple collapse, which may be attended with convulsions.

Case 72.—In March, 1865, I was consulted by a retired officer, aged forty-five, single, who had "lived very hard." He had had gonorrhœa, syphilis, and delirium tremens. He now suffered from what he called "sciatica" in the right leg, but which on closer examination turned out to be lightning pains. He had great difficulty in walking, with numbness in the legs, weakness of the bladder, bowels and sexual organs, and numbness in the hands, more especially in the right, which also showed signs of ataxy. A month after I had first seen him, he had one of his habitual drinking bouts, which led very nearly to another attack of delirium. He heard voices, saw visions, and was "frightened to death," although perfectly conscious and able to converse. This yielded to morphia, but the patient now lost ground rapidly. In June of the same year, he was taken to Wildbad, in Germany, and died there, quite suddenly, in a severe fit of convulsions.

Diseases of the heart and sclerotic or sclero-atheromatous endo-aortitis, occur not unfrequently towards the end of the malady; and Berger and Rosenbach[1] have seen seven cases of coincidence of tabes with aortic insufficiency. These authors consider that there is a relationship between the two conditions, while Vulpian leaves this an open question, and seems rather inclined to think that it is a mere coincidence. Letulle and Grasset have collected a number of cases, showing that all kinds of heart disease may occur in the terminal stage of tabes, and that they do not in general cause much trouble at first, but become pronounced in their effects later on, and occasionally lead to a fatal result by syncope.

Teissier[2] who has quite recently investigated the same subject, is led to the conclusion that tabes and atheromatous degeneration of the aortic valves are generally found together, and considers them identical in a pathological point of view. There is endo-arteritis of the arterioles, and sclero-fibrous degeneration of the blood-vessels is the result. In most cases this affection of the aortic valves may not be recognised in the living; while the autopsy shows small perforations of the aortic valves which have nothing to do with senile decay, as they are found at such an early age as twenty-five. The same morbid changes are also discovered in multiple sclerosis, epilepsy, paralysis agitans, etc.

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[1] "Berliner klinische Wochenschrift," p. 402, 1879.

[2] "Lyon Médical," 1884, No. 6.

As patients linger on the road to dissolution, paralysis invades first one leg and then the other. Permanent contractions of the affected limbs are then gradually established, showing that the disease has crept on to the lateral columns of the cord; or there is great muscular wasting, owing to sclerosis of the anterior horns of the central grey matter. Sometimes a kind of sub-acute poliomyelitis is observed, which entails speedy loss of farado-muscular contractility, and destruction of the muscular substance. Sensibility is now almost entirely in abeyance, but may still be roused for a time by energetic faradisation of the skin. Bedsores form at the sacrum and heels; the paralysed bladder becomes affected by ulceration and gangrene; inflammation may then spread to the pelvis of the kidneys, and the patient is carried off by pyelo-nephritis. Phthisis, heart and aortic disease, obstruction of the bowels, and acute meningitis, may all in their turn put an end to the sufferings of the patient, who is at last glad to die and be released from a life which is in truth worse than death. Only those who are highly gifted with intellectual powers, and have preserved these undecayed to the end, may still look upon their own condition at this stage with a kind of grim humour.

Thus the German poet Heine, who died of tabes in Paris in 1856, after the most frightful tortures, says in his introduction to that wonderful work, "Romanzero," written by a hand which was nearly paralysed, while ptosis prevented him from seeing what he had written:—

"Do I really exist? My body is so shrunken that I am hardly anything but a voice; in my mattress-grave in the noisy city, I hear early and late nothing but the rolling of vehicles, hammering, quarrelling, and piano-strumming. A grave without repose, death without the privileges of the dead, who at least need not spend any money, nor write letters or books—that is indeed a pitiful condition. Long ago the measure has been taken for my coffin and my obituary; but I die so slowly that the process is tedious for myself as well as my friends. What avails me that enthusiastic youths and maidens crown my marble bust with laurel, when the withered hands of an aged hag are putting blisters behind my ears? What avails me the incense of the roses of Shiraz, when in the wearisome loneliness of my sick room I get no perfume but the smell of hot towels? But patience; everything has an end! You will one day find the booth closed where the puppet-show of my humour has so often delighted you."

CHAPTER VII.

THE DIAGNOSIS OF TABES SPINALIS.

THE symptoms of this disease have been so fully described in the preceding chapter that our remarks on the differential diagnosis of tabes from other diseases may be brief. More especially the second or ataxic stage presents such striking features, that a competent observer will often recognise it at a glance ; while on the other hand, in the initial, and again in the terminal stage, of the malady, it will occasionally require all the diagnostic resources at our disposal for recognising the true nature of the disease.

1st. *The initial or pre-ataxic stage* often resembles in some points that form of functional derangement of the spinal cord which is known as spinal debility or *neurasthenia*. In both affections there may be a general feeling of lassitude and want of energy, particularly in the motor sphere ; the patient has a difficulty in walking or standing for any length of time, and is easily knocked up after trifling efforts. The neurasthenic frequently complains of an aching pain in the legs after exertion ; but this is not nearly so violent as the lightning-pains of tabes, and more apt to be continuous than intermittent.

In neurasthenia the back is often in a state of hyperæsthesia ; pressure on, and percussion of, some of the spinous processes of the vertebræ cause intense pain, and may give rise to exaggerated reflexes in the neighbouring spinal muscles. Schuster[1] states that while there may be, in the first stage of tabes, short shoots of pain in the back, there is no tenderness on pressure; and that tabes may thereby be distinguished from neurasthenia, where there is much pain in the neck, back and shoulders, and which is increased by pressure. There are, however, exceptions to this rule; and the case related on p. 200 shows that there may be a high degree of hyperæsthesia in the back, with tenderness on pressure, in the first stage of tabes. In both diseases which we are now considering, there may be a feeling of coldness and numbness in the hands and feet; the sexual power may be diminished; nocturnal emissions may be frequent; sexual indulgence, even to a very moderate extent, may lead to increased debility; and a degree of anæmia may be present. Finally, there may be in both intense depression of spirits, and fear of an impending severe illness. On the other hand, however, we find that there are scarcely any objective symptoms in neurasthenia; the various tests which we use in the diagnosis of tabes are gone through with the most satisfactory results; there is no loss or exaggeration of the patellar reflexes; no incontinence of the urine or fæces; no sign of optic atrophy; no crises of any sort; the pulse is rarely accelerated. The pupils may be habitually large, but respond perfectly to light. The absence of all objective symptoms pointing to tabes is therefore in general sufficient for a diagnosis; and it is a mistake to suppose that neurasthenia ever merges into tabes, as has been frequently asserted. The two conditions are essentially distinct from the beginning, and remain so during their entire further progress, whatever may be their duration.

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[1] "Diagnostik der Rückenmarkskrankheiten," 2te Auflage, p. 73. Berlin, 1884.

With *hysteria*, as generally understood, tabes has no chance of being confounded, but that particular affection which is so common in young women, and known as *irritable spine*, has certainly some features in common with tabes. Irritable spine is no doubt frequently found in the selfish victims of hysteria; but it also occurs in the higher class of neurotics, who may be full of energy, and only too eager to get well and take an active share in the toils and pleasures of life. The principal symptom is backache, chiefly between the shoulders and the nape of the neck, combined with extreme hyperæsthesia, so that the least touch is unbearable; and as the pain and hyperæsthesia are apt to be increased by the most trifling exertion, the patient is necessarily reduced to the condition of an invalid. In addition to this there is a great degree of motor debility, just as in neurasthenia and some forms of the initial stage of tabes. Other symptoms vary according to the portion of the spine which is chiefly affected. Where the cervical part suffers more particularly, there is in addition to the backache, vertigo, pain in the head, more especially the back of it, sleeplessness, hiccough, retching, vomiting, palpitations of the heart, and great difficulty in using the arms and hands. The patient is unable to dress herself without assistance, cannot play the piano, embroider, or write letters. If the dorsal portion of the spine is more affected, there is gastralgia, nausea, vomiting, distension of the abdomen, and asthma; and where the lumbar part suffers, shooting pains through the legs, impossibility to walk, obstinate coldness of the feet, difficulties in urination, defæcation and menstruation are the chief symptoms. The patients are therefore often exceedingly ill, yet there are few objective signs such as we meet with in the first stage of tabes; and the tendon reflexes are never absent, but almost invariably *greatly exaggerated.* This latter sign speaks decisively against tabes.

Rheumatism, *gout*, and *rheumatic gout*, are frequently believed to be the cause of the lightning-pains of tabes. An eminent member of the profession in London, who is well advanced in the second stage of tabes, always speaks of his complaint as "gout." Attacks of lightning-pains are set down as paroxysms of "gout" induced by incautiously partaking of his excellent Burgundy and port wine; and the habitual difficulty in walking is ascribed to gouty deposits in his hips, knees and ankles. Most lay-patients describe their lightning-pains as "rheumatic," and consider that their symptoms are owing to chronic rheumatism. Tabes can never be confounded with rheumatic fever; but some forms of subacute and chronic rheumatism may occasionally resemble it. The rheumatic pain, however, is essentially different from the lightning pain. It is much more constant, not so severe, and more apt to be increased by movement. The regular attack of gout, again, cannot be confounded with tabes, as in the latter there is no acute inflammation of the small joints; but some forms of irregular gout may occasionally lead the unwary astray. There may be in that affection acute gastralgia, resembling a gastric crisis, with intense spasmodic pain in the epigastrium, and bilious vomiting, and some degree of collapse. There may also be a kind of intestinal colic, resembling the intestinal crisis (p. 210); a feeling of constriction of the chest, præcordial anxiety, and asthmatic attacks not unlike the laryngeal crises (p. 186). The history of the case, the presence of gouty concretions, and the absence of Westphal's symptom, will, however, even in apparently anomalous cases, be sufficient to establish a correct diagnosis.

The *arthropathy* of tabes cannot well be confounded with rheumatic gout. We have seen (p. 216) that in the former there is a sudden swelling in a joint, with effusion of synovia, and unaccompanied by pain, redness, or heat; the principal seat of the affection being the knee or shoulder-joint. In rheumatic gout on the other hand, the evolution of the arthropathy is slow; there is only a small quantity of liquid effused, and the principal seat of the affection is in the joints of the fingers and toes, and the hip-joint.

Patients suffering from *diabetes*, on the other hand, may present numerous symptoms resembling those which are observed in the initial period of tabes. The earliest, most frequent, and, diagnostically speaking, most important symptom in the sphere of the nervous system which occurs in diabetes, is one which we also know as a symptom of tabes (p. 205) and spinal neurasthenia, viz., a sensation of fatigue, lassitude, and utter want of muscular energy. This does not depend on the emaciation of the muscles, which is one of the later symptoms of the malady; but comes on, without any apparent cause, either in the lower extremities or in the loins, and may be severe enough to raise a suspicion of spinal disease. There is difficulty in walking; the movements are slow, awkward, and without vigour. This lack of power may come on quite suddenly, for instance after a slight accident, and is possibly owing to defective muscular nutrition from the blood being saccharine.

Various forms of paralysis are also observed in diabetes, which may be local, partial, and incomplete; or there may be regular hemiplegia. The latter may come on suddenly, with apoplexy, and may get well in a few days, just as occurs in tabes (p. 194), and there may be a repetition of these symptoms at some subsequent period. In other cases the apoplexy proves fatal, or there may be simple loss of consciousness without subsequent paralysis, or a bad attack of vertigo. Again, paralysis may come on without being preceded by apoplexy; and hemiplegia of one side may be combined with monoplegia of the other. Monoplegia is indeed so frequent in the course of diabetes that it is always incumbent on the practitioner to examine the urine for sugar in these cases. Palsies of this kind may be confined to a limb, or part of a limb; to a single muscle, or a small group of muscles in the face; and they affect frequently the tongue and the muscles moving the eyeball. They may be quite transitory, indeed last for a few hours only, and are often incomplete.

Difficulty in speaking may be either [illegible] asic or the anarthric kind, or be simply owing to general debility combined with dryness of the tongue. Sometimes, there is a more or less complete loss of the memory for words. A temporary kind of aphonia is probably owing to transitory paralysis of the muscles of the larynx. There are not as yet any very conclusive observations on palsies of the muscles of the eye, although Kiwatkowski has recorded a case of paralysis of the fourth nerve in a diabetic patient, and the rectus externus has been found paralysed under similar conditions.

Another interesting symptom in the motor sphere in diabetes is a tottering gait, especially in the dark. This may be combined with "pins and needles" in the lower extremities, just as in tabes. Indeed, one cannot help being struck by the close similarity of many of these symptoms to those of locomotor ataxy.

A symptom, however, which is frequent in diabetes, and absent in tabes, is *cramp in the legs*, which occurs chiefly at night, and leads to *sleeplessness*. The latter, however, may also occur in diabetes simply from the nutrition of the brain being impaired through the saccharine blood ; indeed, if insomnia appears without any apparent cause, diabetes may be suspected. In tabes, insomnia occurs generally only during attacks of lightning-pains or crises of any description.

On the other hand, we find in diabetes symptoms which again closely resemble those of tabes, viz. :—In the sphere of sensibility local areas of anæsthesia and analgesia, so that hairs may be pulled out without causing any pain to the patient. More frequently there are complaints of tingling, tightness, cold, heat, and numbness, especially in one or both lower extremities or in the sexual organs. Like the tabid patient, the diabetic is exceedingly sensitive to cold. Tactile sensibility may vanish, so that the patient is unable to hold a pin between his fingers without looking at it. Some patients lose the proper sensation of the ground on which they are walking. Pain in the joints, the loins, the haunches, and the back is common; and it seems particularly to invade the neck, where it is felt as a burn or the bite of a dog, and is combined with stiffness of the muscles. This rigidity extends occasionally from the back of the head down to the sacrum. There may also be headache, pressure on the top of the head, and various forms of neuralgia, more especially symmetrical and obstinate sciatica. Lightning-pains, similar to those of tabes, have likewise been observed, and may, if combined with difficulty in standing, plantar anæsthesia, and areas of hyperæsthesia, lead a hasty observer to the diagnosis of tabes.

Sexual desire is generally in abeyance. In men there is frigidity and impotency; in women, actual repugnance to connection. Certain neuroses, such as asthma, angina pectoris, etc., may resemble the laryngeal crises which are known to occur in tabes. Deafness is more frequent in the diabetic than in other persons, and seems sometimes purely nervous, while in other cases it is owing to lesions of the middle ear. Anorexia and perversion of the sense of smell have also been observed.

Certain peculiar nutritive disturbances which we have seen to occur in tabes (p. 218) may also occur in diabetes, more especially perforating ulcer of the foot, bed-sores, localised sweating, and muscular atrophy.

An additional element of uncertainty in these cases is that sugar may be found in the urine of the tabid (p. 263); while on the other hand Pavy has shown that the palsies and other nervous symptoms of diabetes may occur some time before the diabetes appears, or just when, in consequence of treatment or otherwise, the sugar disappears from the urine, or after it has been completely absent from it for several months consecutively. The other symptoms of diabetes, such as hunger, thirst, polyuria, emaciation, etc., may under such circumstances not be very marked, and where the nervous symptoms which have been described are very prominent, as is chiefly the case in those having inherited the neurotic constitution, diabetes and tabes may indeed resemble one another in the closest possible manner; so closely indeed that occasionally a decision would be actually impossible without our sheet-anchor for the diagnosis of tabes, viz., Westphal's and Argyll-Robertson's symptoms, which never occur in diabetes, and therefore are of the utmost value when we are called upon to decide the nature of such excessively obscure and doubtful cases.

Amblyopia and amaurosis also occur in the course of diabetes, adding another symptom common to both diseases. Here, however, the ophthalmoscope steps in, showing that in the amblyopia of the earlier stages of diabetes, the fundus of the eye is normal, or that there is at most a slight congestion of the optic disc. The amblyopia of diabetes is indeed owing to paresis of accommodation, which is no doubt caused by the saccharine state of the blood. It may, therefore, be compared with uræmic amaurosis, where occasionally likewise no ophthalmoscopic changes are observable. In general the two eyes are unequally affected; all objects appear to the patient in a kind of yellowish haze; and this is worse after meals. A severer form of true amaurosis may come on towards the end. This is owing to glycosuric retinitis, and resembles albuminuric retinitis, and the changes in the retina seen in pernicious anæmia. Hæmorrhage occurs almost invariably, and this may lead to secondary parenchymatous retinitis. Simple atrophy of the optic nerve may, however, likewise exist, and thus render the diagnosis even more doubtful. The commonest cause of defect of sight in diabetes, however, is cataract, which does not occur in tabes, and therefore may be utilised for diagnostic purposes.

The patient whose case (No. 55) is described on p. 195, and who suffered from tabes combined with glycosuria, had been pronounced to suffer from diabetes by a distinguished physician, and on this account been sent to Neuenahr—a spa utterly unsuited for his condition. This shows the practical importance of the diagnostic tests between tabes and diabetes which I have mentioned.

Amblyopia and amaurosis from *optic atrophy* in tabes may be distinguished from impairment or loss of vision through optic neuritis, such as we see it in tumour of the brain, meningitis, abscess, and softening of the brain from embolism or thrombosis, etc., chiefly by the evidence obtained with the aid of the ophthalmoscope. The ophthalmoscopic signs of optic atrophy (p. 172) are chiefly pallor and excavation of the disc, the edge of which appears extremely sharp; while in optic neuritis, on the contrary, we have increased redness, swelling and cloudiness of the disc, the edge of which is blurred and imperceptible, the centre being darker than the periphery, while the veins are enlarged and the arteries narrowed. The other clinical symptoms of the two affections do not present such a striking contrast. It is true that in optic atrophy one eye always commences to fail before the other, and that optic neuritis is often at once bilateral; yet the affection may also commence in one eye in optic neuritis. Vision becomes impaired or lost in both diseases, and in both there is limitation of the visual field, and achromatopsy, red and green being lost before blue and yellow. In general, however, optic neuritis is much more rapidly destructive of sight than atrophy, for blindness may occur in the former within two or three days, while it will take at least as many months or years before sight completely fails from optic atrophy. Blindness, however, never comes on instantaneously from optic neuritis, as it will do from embolism of the central artery of the retina. From this it will be seen that the ophthalmoscopic signs are by far the most valuable. Charcot relates a case where it was difficult to determine whether a patient was suffering from tabes or from tumour of the occipital lobes of the brain, and where Galezowski was enabled to make a correct diagnosis by means of the ophthalmic mirror. It is right to say that this case occurred at a time when Westphal's symptom was not yet habitually utilised in France, as otherwise no such difficulty could have arisen; for in tumour of the brain, wherever situated, the tendon reflexes are invariably exaggerated, while in tabes they are lost.

Lightning-pains are often confounded with *neuralgia*, more especially when they affect chiefly or exclusively the sphere of a certain nerve or nerve-branch. The pain in both affections may be equally severe, and present the same characters as regards intermittence, etc. A search for other symptoms of tabes, however, will in cases of neuralgia be generally unsuccessful, while in tabes it will result in the discovery of the loss of the knee-jerk, Argyll-Robertson's symptom, etc.

Gastralgia, on which Clifford Allbutt[1] has recently discoursed so eloquently and impetuously, may at first sight closely resemble the gastric crisis. The pain is equally intense, and the attack liable to come on suddenly and without much warning. Yet gastralgia occurs chiefly in young women, and is often preceded and followed by other symptoms of stomach-derangement; while the gastric crisis occurs chiefly in men in the prime of life, is accompanied by other symptoms of tabes, and rarely by continuous derangement of digestion.

Enteralgia, as described by the same observer, may occur as a pure neurosis or as a symptom of gout; and it may closely resemble the *intestinal crisis* of tabes. The severity of the pain is very great in both conditions, for Allbutt's patients also complain of "stabs and racking of a knife," and of such frightful agony that death seems preferable to the torture they endure. The pain of enteralgia is chiefly seated in the right iliac fossa and the umbilicus. Neuralgia of the liver, kidney, rectum, and bladder appears to fall into the same category; and so would neurotic attacks of diarrhœa. The latter occur from nervous causes, and are generally associated with other neuroses, such as migraine, cardio-vascular instability, etc. In all these affections the presence or absence of the knee-jerk will, in the last resort, be of decisive diagnostic importance.

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[1] "On Visceral Neuroses," p. 30. London, 1884.

In those cases of tabes where gastric crises form a prominent symptom, *certain diseases of the stomach* have until quite recently been confounded with the cord-disease; and the diagnosis of ulcer or cancer of the stomach, or of biliary and renal calculi, has been freely made. Hæmatemesis more especially has until lately been almost invariably referred to ulcer or cancer of the stomach, but we know now that it is a symptom which is apt to supervene towards the end of the gastric crisis, and simply through excessive nervous irritation without actual disease of the stomach. Yet there are many points which will help us towards a proper diagnosis of the case. We should remember that *ulcer of the stomach* is much more frequent in women than in men, while in tabes the reverse is the case; that it is more common in advanced age, while tabes is a disease of the prime of life; that the pain of ulcer always comes on after, and in consequence of, taking food, while the gastric crisis of tabes is quite uninfluenced by this; that in ulcer the pain is generally localised in the epigastrium or left hypochondrium, or in a certain portion of the back, and there is tenderness on pressure, while in the gastric crisis the pain shoots over a considerable area, and is not materially increased, but often rather relieved by pressure. Vomiting in ulcer is not common, and almost invariably ceases after the food ingested has been brought up, while in the gastric crisis it is incessant, and continues long after the contents of the stomach and duodenum have been voided. Ulcer is distinguished by its chronic course, and the gastric crisis by an extremely acute occurrence. Finally, in ulcer the patellar reflex is present, and in tabes it is absent.

Perforation of the stomach and bowel, occurring from ulceration, resembles in some respects the gastric crisis: there is intense pain in the abdomen, and vomiting, and it may occur where there has been only little complaint of previous ill-health. But in the gastric crisis the patient is restless and writhes about in bed, while in perforation he keeps perfectly still in the recumbent position, and dreads the slightest movement; the symptoms of peritonitis soon become developed, and are then unmistakeable. The passage of *biliary and renal calculi* is sometimes ushered in by symptoms exactly resembling the gastric crisis; and in such cases we have occasionally to rely exclusively on Westphal's symptom in our diagnosis of the case. Dilatation or atrophy of the coats of the stomach, on the other hand, are on account of their essentially chronic course, not likely to be confounded with gastric crisis.

Cancer of the stomach occurs mostly in advanced age; there are almost invariably long-standing symptoms of indigestion, with pain in the stomach, whether the latter be full or empty, and great loss of appetite, emaciation, and a greenish or jaundiced complexion. The symptoms do not vary much from day to day, but it should be remembered that the first symptom of gastric cancer may be hæmatemesis. Westphal's symptom will in any doubtful case clear up the nature of the affection.

The urinary troubles from which many tabid patients suffer are in practice frequently referred to *local diseases of the urethra or bladder*. The aid of the bougie or catheter is called in, and the patient is frequently made much worse by this local interference. There can be no doubt that the tabid are extremely vulnerable; and if the symptoms described on p. 206 be present, the surgeon should never omit to look to the knee-jerk. If it is found to be absent, local instrumental interference can in most cases do no good, and may do a great deal of harm. I am inclined to believe that, at least in a certain number of those cases of fatal catheterism which Sir Andrew Clark recently brought before the Medical Society of London, incipient tabes may have been present. It is true that it was stated that no post-mortem change was discovered in any important organ; but it is at least possible that the spinal cord may *not* have been examined with all due care, including more especially hardening and staining. In all cases of *impotency*, *spermatorrhœa*, and *satyriasis* which may come under our care in practice, a special search for other symptoms of tabes should be instituted, as these symptoms acquire an entirely different significance when owing to tabes, from what they have if due to local disorders of the sexual organs and other conditions.

Attacks of hemiplegia and aphasia, which we have seen to occur in tabes (p. 194), may be puzzling unless the history of the case be known. A mistake in diagnosis will here often lead to a wrong prognosis, as the hemiplegia and aphasia of tabes are almost invariably temporary, and not owing to any structural lesion of the brain. The patellar tendon reflex is here again of the greatest semiotic importance, and its loss will show the nature of the case more definitely than any other symptom.

2. *The second or ataxic stage of tabes* shows such highly characteristic features at first sight that the disease can hardly be confounded with any other at this period by a competent observer.

It has been stated that tabes has been occasionally confounded with *chorea*; and it is quite true, that "muscular madness" occurs at a certain period of the ataxic stage of tabes (p. 232), as well as in chorea. Nevertheless it seems almost impossible to mistake either of these affections for the other. Chorea occurs chiefly between five and fifteen years of age, and much more frequently in the female than in the male sex; moreover the choreic twitches occur spontaneously, and in an entirely different manner from what is seen in ataxy. It seems equally impossible to mistake tabes for *paralysis agitans*, in which the chief symptoms are tremor during rest and loss of muscular power, while pain is ever absent, and the tendon reflexes are normal. Other forms of *paralysis*, owing either to cerebral or spinal disease, are distinguished by loss of muscular force, which latter in the second stage of tabes is almost invariably normal. In ordinary hemiplegia and in transverse myelitis the patellar reflex is increased, unless, what is rarely the case, the lumbar enlargement should happen to be affected.

Multiple sclerosis may be distinguished from tabes chiefly by the peculiar rhythmic tremor which is seen when voluntary movements of a certain extent are made or intended to be made; by muscular rigidity; and increase of the tendon reflexes. *Disease of the cerebellum*, such as tumour, softening, etc., causes a staggering or "reeling" gait, like that of a drunken man, with an impulse to fall backward, forwards, or to the side; the patient does not use his eyes as crutches; there is generally severe continuous pain in the back of the head, which often extends from thence in gradually diminishing intensity to the other parts of the head; vomiting, optic neuritis, vertigo even in the horizontal position; no pain, anæsthesia, paræsthesia or analgesia in the limbs; and no signs of ataxy of the upper or lower extremities in the horizontal position.

Chronic spinal meningitis is often present as a complication in the second stage of tabes (pp. 14, 42), and may be suspected when there is much pain and stiffness in the back, with tenderness on pressure; primary spinal meningitis, however, cannot be confounded with tabes, as it causes paralysis with muscular rigidity, and no ataxy.

3. *The terminal stage of tabes* may possibly be confounded with the terminal stage of a number of chronic affections of the nervous centres. When complete paralysis of the bladder, bowels, and lower extremities has been established, when bed-sores have formed on the sacrum, the heels, and

other parts, when the patient is blind, deaf, emaciated and helpless to the last degree, then we have indeed to deal more with approaching dissolution of the nervous centres than with any definite localised affection. *General paralysis of the insane* may be combined with tabes in various ways, so that occasionally the cerebral and in other cases the spinal symptoms predominate ; yet in more definite and less diffuse lesions the diagnosis cannot be doubtful. In many cases of tabes the mind remains clear and unclouded to the last ; there are no delusions ; speech is rarely much, if at all, affected, and the pupils are equal. In general paralysis, on the other hand, the speech is always affected ; the pupils are generally unequal ; there is tremor of the tongue and facial muscles ; failure of memory, delusions, and imbecility. In cases where general paralysis becomes complicated with tabes, or *vice versâ*, the patellar reflexes are absent ; but where the disease is entirely or almost entirely cerebral, these reflexes are either normal or more frequently exaggerated.

CHAPTER VIII.

THE PROGNOSIS OF TABES SPINALIS.

As late as 1851, Romberg wrote that there was no hope for patients of this class ; that sentence of death had been passed upon them, and that it was but common humanity to inform them that therapeutic interference could only injure. Trousseau, in 1862, said that the prognosis was of extreme gravity, and that the multiplicity of the remedies used for combating the disease testified to their uselessness. Duchenne, in 1877, gave a somewhat more hopeful view, and laid stress on the importance of an early recognition and persevering treatment of the complaint. Vulpian, in 1879, considered the prognosis serious ; for although the disease was sometimes arrested and for a time even improved, it had in general a progressive tendency, and the patient must die of it unless he were carried off by some intercurrent affection ; while during life he was tortured by pain, sickness, the horrors connected with the affections of the bladder and bowels, blind, deaf, unable to earn his living, and entirely dependent upon others. In 1883, Leyden said that we were not only unable to cause the lesion of tabes to disappear, but that there was hardly any prospect of our ever attaining this end in future.

It appears to me, that since the discovery of the part which syphilis plays in the etiology of tabes, and likewise since the discovery of a symptom which allows us to recognise the latter malady in the very beginning, the aspect of this question has become entirely changed. We know now that any one who has at any time suffered from constitutional syphilis may, at a period more or less remote from the infecting sore and the secondary manifestations, become affected with tabes. The period of latency varies, according to the cases which have been reported in this volume, from twelve months (p. 95) to twenty-seven years (p. 121); and a patient, therefore, who has once been syphilitic, cannot consider himself safe from the liability to tabes for the better part of his life afterwards. This fact, which can no longer be disputed, involves a serious responsibility on the practitioner who has to treat a patient affected with venereal disease. We have seen that more especially those persons who suffer from comparatively mild secondary symptoms, and who are therefore insufficiently treated, become later in life the victims of tabes (p. 84); and this disease would, probably, never have become developed in them if the syphilitic taint had been energetically and perseveringly combated at the time of its first manifestations.

When the venerable Ricord attended the meeting of the British Medical Association at Birmingham, in 1873, he delivered an address in which he stated as the outcome of his unrivalled experience in the treatment of syphilis, that a patient who had been properly treated for two years subsequently to his having contracted the infecting sore was cured, and might consider himself free from any liability to further secondary or tertiary manifestations.

I would, therefore, say to the practitioner: Ponder well over Ricord's teaching: take syphilis well in hand at the earliest stage of it. Do not comfort the patient who comes to you with an infecting sore with speedy prospects of a cure, but tell him plainly that he has become inoculated with an insidious and dangerous poison, which may maim or kill him sooner or later unless he submits to systematic and persevering treatment. Do not rest in your endeavours until you have reason to believe that every germ and vestige of that fearful virus has been thoroughly uprooted and destroyed. Examine those of your patients who have had secondary symptoms, from time to time for the knee-jerk; inquire of them whether they have had temporary double vision or incontinence of urine; examine them for Argyll-Robertson's symptom; and if they should complain of a sensation of a tight rope round the chest, do not at once rush to the diagnosis of dyspepsia and flatulence. Do not think that every severe pain in the legs is owing to rheumatism or sciatica; hunt for the symptoms of tabes in cases of grave laryngeal, gastric, or intestinal troubles, which may have been preceded by syphilis. More especially should you find the knee-jerk absent, take it to be a warning that that terrible malady, tabes, has pushed forward its outposts, and is on the point of invading the system. Set to work, then, at once to obstruct its further progress by every means in your power, and let us thus, by suppressing that most painful disease, locomotor ataxy, add one more triumph to the victories of PREVENTIVE MEDICINE!

I have, then, no hesitation in stating that the prognosis of tabes has, in consequence of the advance of our knowledge of the causes, the early symptoms, and last, not least, our means of treating the disease, become much more favourable in recent years; and I am convinced that when the malady is better known in all its details to the entire profession, and therefore more clearly and readily recognised in the very beginning, the prognosis will become more favourable still as time goes on. In cases where the primary and secondary manifestations of syphilis have been insufficiently treated, the patient should be subjected to a prolonged course of specific treatment. By doing this I have in many cases of this kind, in which the prognosis appeared to be *primâ facie* extremely unfavourable, succeeded in arresting the further progress of the disease, and in preventing any subsequent manifestations of the syphilitic dyscrasia. Some patients, in whom a vast deal of mischief had already been done, have to all appearance permanently recovered; and I am convinced that by far the largest majority of them would never have suffered from syphilitic disease of the nervous centres—which is day by day killing many persons and crippling a good many more—if the primary sore and the earlier secondary symptoms had been treated in that systematic and persevering manner which Ricord has shown to be necessary.

Although we, therefore, look upon cases of tabes no longer as hopeless, we must, nevertheless, not disguise from ourselves the fact that it is one of the most obstinate complaints with which we have to deal in practice; that some of its symptoms, more especially optic atrophy, may possibly be arrested in their further course, but that any actual mischief which has been done in such parts as the retina is irreparable; that the disease is never a mere functional derangement, but at the very beginning one of destruction of tissue; and that the patient is, therefore, even at best, and when his health has been apparently restored, more vulnerable than another whose spinal cord has never suffered, and less able to stand the wear and tear of life than he was previous to the affection.

I now proceed to consider how far the possibility of recovery in tabes is limited by the peculiarities of structure of the affected parts, and of the disease which has invaded them. With regard to the latter, it must always be kept in mind that there is never, at any time, a mere functional or prodromial stage, in which we could assume that we had simply to do with finer molecular changes in the nutrition of the affected parts, which might be easily rectified; but that there is actual sclerosis from the very beginning.

Experiments in animals, more especially by Eichhorst and Schieferdecker, have shown that a division of, or injury to, the cord may to some extent be repaired, more especially where the injury has not been very extensive, and where the animals are young; and that this repair is chiefly seen in the nerve-tubes, and much less, if at all, in the ganglionic cells of the grey matter. The morbid anatomy of the human subject has shown similar results. In patients who have died of transverse myelitis as well as of tabes, newly formed nerve-tubes have been found by the side of wasted fibres, and apparently originating from them; and the persistence of the axis-cylinder, which was formerly believed to be necessary for regeneration of the tube, has been shown not to be indispensable. Ganglionic cells, which are more highly organised than simple nerve-tubes, appear to be incapable of regeneration. The large cells in the anterior cornua of the cord, which perish in infantile paralysis, have been found completely wasted years after the attack of that disease had taken place, and even after considerable recovery of motor power and muscular tonicity had taken place. The nerves of the higher organs of special sense, which have a particularly complex and highly specialised structure, would appear to be *primâ facie* incapable of repair. Indeed, it is found that the rods and cones of the retina and the fibres of the auditory nerve contained in the crista of the semicircular canals of the membranous labyrinth, when once much injured by hæmorrhage, atrophy, or other morbid processes can never recover. The patient whose case (No. 53) is described on p. 176 got perfectly well of all symptoms of tabes, except of the deafness which had been caused in him by an attack of auditory neuritis. With regard to the capacities of the neuroglia in this respect, only little is known; and equally little about any recuperative power of the coats of the blood-vessels which may have been pathologically altered.

Although, therefore, the central nerve-tubes, which are the principal seat of the lesion of tabes, have been shown to be capable of regeneration, it is nevertheless probable that any very extensive reproduction of them is rare, more especially in persons of a somewhat advanced age. The cure of the symptoms of the disease which we may witness clinically has therefore to be attributed to the circumstance that, apart from the perished or incurably diseased fibres, others have been in a state of only commencing degeneration, which latter was not only arrested, but actually repaired; while again others, which would no doubt eventually have succumbed to the same morbid influence, were spared altogether. More especially in recent cases, a more or less considerable number of nerve-tubes is left, which may not only be saved from destruction, but which may be considerably strengthened by the use of proper stimulants. Finally, the "law of substitution" may become to some extent operative, causing healthy fibres in the neighbourhood to perform the work of others which have perished. If, then, we observe clinically that the symptoms of tabes gradually improve and vanish, we must not assume that all the anatomical changes which have taken place in the central nerve-tubes have been repaired; but that there has been improvement in the nutrition of those fibres whose structure was not irretrievably damaged, and vicarious action on the part of neighbouring tracts.

From these premises, then, it appears that, under favourable circumstances, the two principal symptoms of tabes, viz., the lightning-pains, with all their modifications (crises, etc.), and the locomotor and static ataxy may wholly or partly yield to therapeutical measures. The same may be said of the vesical, rectal, and sexual troubles. With regard to the muscular atrophy, which not unfrequently accompanies tabes, less hope may be indulged in, as we have seen that ganglionic cells, when once destroyed, are never repaired. Yet we must not lose sight of the fact that, where only some portions of these cells remain physiologically active, they may still be able to exert a powerful influence on nutrition and tonicity. Adamkiewicz[1] has recently seen a case in which the pressure of a tumour in the left side of the cervical enlargement of the cord had caused the ganglionic cells of the central grey matter to be reduced to the thirty-second part of their original size, and yet no wasting of the muscles corresponding to the injured parts had taken place. Finally, the optic and auditory atrophy of tabes may under favourable circumstances be arrested in their further course; but from the highly specialised structure of the involved nerves, a repair of destroyed tissue cannot be expected.

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[1] "Wiener Medicinische Presse," No. 9, 1883.

The prognosis of tabes can therefore, on the whole, at present not be considered so dismal as most of the authorities quoted above would lead us to believe. We possess in mercury and iodide of potassium true specifics for the dyscrasia which in the enormous majority of cases leads to tabes; and we possess likewise in electricity, nitrate of silver, and ergot of rye, remedies by which the nutrition and functional activity of the central nerve-tubes may be wonderfully promoted and stimulated. Much must always depend upon the time when the patient comes under treatment, upon his obedience to our rules, upon his age and general stamina, as well as upon a variety of more individual circumstances. While we cannot hope to cure the fully-developed malady, we know that at least in a certain proportion of cases where the disease was not very far advanced, very great improvement, and, in some, more or less complete recovery, has taken place.

The patient whose case is described on p. 264, and who was well advanced in the third stage of tabes, made wonderful strides towards recovery under proper treatment, until an unfortunate relapse into dissipation arrested his progress, and killed him. Patients who give themselves unreservedly and absolutely up to our rules and prescriptions, and consent to live by command, even in the smallest details, while they are under our care, have an infinitely better chance than those who criticise our advice or refuse to obey us implicitly. Then the external circumstances of the patient are of the greatest importance. Out-patients of hospitals have no chance of recovery; the poor, or even those who are not able to have everything that is necessary for them, should be admitted as in-patients at the earliest possible period. Those born with the silver-spoon in their mouths are, here as elsewhere, the favourites in the race to get well. Temperament and individual constitution are likewise of great influence: the rake will succumb more quickly than the philosopher. Of two of the most marked cases of tabes which I ever saw, one occurred in a peer, who being of slight physique, and calm and fastidious by temperament, surrounded himself with everything that may adorn life, and found consolation for his infirmity in the arts and literature. He lived to the age of sixty, after having been subject to the complaint for upwards of thirty years, and having hardly ever experienced really severe suffering. The other case was that of a hot-tempered Irishman, who, after having made a fortune in Australia, returned to England to spend his life in the wildest excitement, intrigues, and debauchery of every description. He died within four years from the outbreak of the malady, after having undergone the most frightful tortures which man may be called upon to endure, and having seen an originally herculean constitution undermined and wasted in a comparatively short time.

Generally speaking, the natural course and evolution of tabes are slow. A number of years will mostly elapse, even without active treatment, before the malady becomes fully developed; and there is on the whole therefore not a great tendency on the part of it to shorten life. Many persons suffering from it reach a good age; and the disease often remains stationary for many months. Nevertheless, patients may die in the first stage, more especially in consequence of the various crises to which they are subject, and any of which may terminate fatally. Others show, some time after having been affected with tabes, great loss of resistance to injurious influences, and succumb to bronchitis, pulmonary congestion, and similar affections which, otherwise, only rarely carry off men in the prime of life, when placed in favourable circumstances. The patients also seem to have a particular tendency to catch typhoid fever and similar infectious or contagious diseases ; and their chances of surviving for any length of time, are therefore, on the whole, more precarious than those of others. It will, however, be imprudent even in the last stage, when the patient is hardly more than a living skeleton, to predict a fatal result within a short time, as in some cases toughness and tenacity are seen in place of vulnerability; and although the patient may be said to be dying by inches, yet the process of dissolution may be at the last surprisingly slow, and devoted and intelligent nursing will occasionally rescue the patient from the very jaws of death.

CHAPTER IX.

THE TREATMENT OF TABES SPINALIS.

IF any real success is to be achieved, the treatment of tabes must be persevered with systematically for many months, and in some cases for years. *Rest* is of great importance, and we must endeavour to arrange all conditions of life as favourably as possible for the patient. I am not a partisan of absolute rest, either mental or physical. Hammond mentions a case where a man suffering from tabes was obliged to keep to his bed for twelve months consecutively, on account of a mismanaged fracture of the thigh-bone, and who, when he got up, was found to be quite free from ataxy. This I believe to be an exceptional occurrence, as complete rest for a considerable time generally makes the patient more lame than he was before, and also depresses the vital powers considerably. I therefore, even in those cases where walking is very troublesome, encourage the patient to take some gentle exercise, as far as is possible, several times a day.

Excesses of every description must be strictly prohibited. Indulgence of the sexual desire is more particularly baneful, and alcoholic intemperance hardly less so. The patient whose case is described on p. 264 was, although in the third stage of the disease, improving in every respect under treatment, when the effects of a drinking-bout carried him off in a short time.

In the following instance, the sexual act proved very injurious :—

Case 73.—In September, 1882, Dr. Grasemann requested me to see a manufacturer, aged fifty-two, married, who had for the last four years complained of "neuralgia" in the left arm, chiefly in the neighbourhood of the deltoid muscle, with a "rheumatic" swelling in the left thumb. The case looked at first sight like one of rheumatic gout; but on closer inquiry it turned out that the patient had had syphilis badly twenty years ago; that the knee-jerk was gone in both sides; that there was not only "neuralgia," but also anæsthesia and analgesia in the left arm; that the patient had occasionally had shooting-pains in the right arm and right leg, not of extreme violence, but nevertheless of a severe kind; that his bladder was very sluggish, and his sexual power diminished. On one occasion lately, when he had had intercourse, it was of an unsatisfactory character, and immediately afterwards he felt as if he were choking or fainting, and suffered from such an amount of prostration that he was unable to leave his bed for two days. At the same time all the other symptoms from which he had suffered had become worse.

Over-exertion on a single occasion may hurry the patient almost immediately from the second into the third stage of tabes, as was the case in the following instance:—

Case 74.—A commercial traveller, aged forty, single, was admitted into the hospital under my care in January, 1878. He had exceeded in drinking and sexual indulgence; had had delirium tremens five years ago, and chancre, gonorrhœa and stricture before then. He had also suffered a good deal from exposure in the Tropics. Two years ago he first felt a difficulty in walking, and was then admitted into the hospital at Buenos Ayres, where he remained for three months, and got better and worse, off and on. At present there is ptosis of the left eyelid and paralysis of the rectus internus and obliquus superior of the left eye. There is no sign of optic atrophy, and no further symptom in the upper portion of the body. Lower down he has numbness in the hips, thighs, legs, and feet, more especially the latter; lightning-pains chiefly in the right leg and foot, but occasionally likewise in the left. The "shoots" last for a few seconds, after which he is quiet for an hour or so; then there is another attack, etc. He feels the ground as soft as velvet. Two months ago he was still able to walk three miles at a stretch, when one day, on crossing a street, he had to run fast in order to avoid a cab going over him. This seemed to give him a strain in the back, and ever since he has been almost paralysed. He is now utterly unable to walk, even with assistance, although he has little difficulty in crossing one leg over the other, and in moving his legs in bed. He can only stand when supported on both sides, and would fall at once if let go. When standing in this fashion, he cannot take his eyes off his feet. The bladder is weak and sluggish, but does not give him much trouble; the bowels are regular; the sexual power is diminished, but he has still desire, and erections and emissions of semen in his sleep. In this case the patient "jumped," as it were, from the second into the third stage, as he became completely helpless almost directly after running a few steps.

Smoking does not seem so injurious, and a cigarette or a mild cigar may be occasionally allowed to those who are fond of this pastime. Exposure to cold, on the other hand, is most injurious (p. 99), and should be carefully avoided.

The diet must be nutritious and easily digestible. Mutton, either roast or boiled, poultry, and the white kinds of fish, such as sole, whiting, and cod, are better than the heavier meats or salmon. Tea and coffee should be taken in strict moderation; of wines, only claret and the higher class of red Hungarian wines are to be allowed. These may be suitably mixed with Salutaris or Apollinaris water. Spirits, sherry, port wine, the heavier Burgundies such as Chambertin, champagne, and bottled beer, should be avoided altogether. Where symptoms of general debility and impaired nutrition are prominent, phosphorus and cod-liver oil, or malt extract, are useful. Phosphorus may be given either in the form of the French pearl, which is stated to contain 1-32 part of a grain, but probably contains only half that quantity; or as phosphide of zinc, made up into a pill with sugar of milk and glycerine of tragacanth, the dose of this being a quarter of a grain.

The treatment of the *syphilitic dyscrasia* is of paramount importance, and most successful when carried out in the first stage of the malady. Mercury is here our sheet-anchor, and may be given either as perchloride or biniodide, or by inunction. The latter is by far the most effective way of administering the medicine; and in carrying it out I greatly prefer the oleate of mercury to the dirty and uncertain blue ointment of the Pharmacopœia, in which the metal is rubbed up with lard and suet. In the oleate sixty grains of the red oxide of mercury are to be dissolved in ten drachms of oleic acid, forming a ten per cent. solution, of which as much as a whole or half a teaspoonful should be applied at bedtime with a brush, and then covered with a silk handkerchief, or rubbed by the patient himself into the skin for a few minutes. The inner surfaces of the thighs and arms are the best places, as absorption from there is most active. This application rarely causes much irritation; but occasionally an ecthymatous rash is produced, which, however, subsides quickly, and may, if necessary, be treated by astringent lotions.

Subcutaneous injection of the perchloride of mercury is a quick, although rather painful, mode of obtaining the effects of the metal without disordering the stomach. The initial dose should be the twenty-fourth part of a grain of the perchloride dissolved in fifteen minims of water. This mode of treatment can only be carried out in stout persons, as the medicine has to be deeply injected into the cellular tissue of the buttocks. In thin persons such an injection would cause a considerable degree of irritation. If no reaction follows the injection of the dose mentioned, it may be gradually increased to the sixteenth or twelfth and even sixth part of a grain, and be repeated daily or every other day. In case the twenty-fourth part of a grain should cause irritation, the dose may be further diminished. As much as half a grain has occasionally been injected, but was found to produce symptoms of mercurial intoxication. Liégeois has recommended the injection of morphia, together with the sublimate, the formula being—

℞ Hydrargyri perchloridi	gr. iii.
Morph. hydrochlor.	gr. jss.
Aq. dest.	℥ iii.

Misc. fiat injectio hypodermica. S. Dose 15 minims (1-32 grain of the perchloride).

Other preparations of mercury recommended for injection are the perchloride of mercury and sodium (Stern), the albuminate of mercury (v. Bamberger) and the peptonate of the metal (Friedlænder). None of them, however, seem to possess any decided advantage over the perchloride.

There is no necessity for causing salivation. If the gums should be slightly touched, and the mouth be uncomfortable, a gargle of the glycerinum boracis in elder-flower water is an excellent application.

I generally combine iodide of potassium with mercury in the beginning of the treatment; and this may be given in doses varying from fifteen to one hundred and twenty grains per diem. Sometimes it is found that the larger doses agree better than the smaller ones; but where the stomach refuses to take the drug easily, ammonia, arsenic, dilute hydrocyanic acid, and similar correctives must be added.

In some cases the iodide of sodium acts better and more pleasantly than the potassium salt; but the latter is in the majority of cases by far the most effective of the two.

Increases in the dose of the iodide must be made, more especially in cases which progress unfavourably, and where, after a short improvement in the symptoms, a somewhat rapid deterioration takes place. This has been occasionally assumed to forbid the further exhibition of the drug, with the result that the patient has become hopelessly diseased. Practitioners are still, as a rule, afraid of giving large doses of this medicine, and disinclined to continue it for any length of time. It is, however, impossible to cure the syphilitic dyscrasia with five-grain doses of the drug given twice a day for a fortnight. The time during which mercury and iodide of potassium should be continued, and the doses in which they are to be given, must depend upon the individual aspect of the case.

In the second and third stage, the effects of the specific treatment are not nearly as marked as in the first. Nevertheless, it is our duty to submit the patient to it, unless, indeed, his system has already become saturated with either mercury or iodide of potassium. In this latter case, and also where there is no syphilitic history, we have to resort to the use of ergot of rye and nitrate of silver.

If either of the drugs mentioned or the application of electricity prove useful in tabes, after the specific treatment has failed to do good, this is no proof whatever for assuming that the affection was after all not owing to syphilis. It is a matter of daily observation that maladies or symptoms are caused in the first instance by specific blood-poisons, such as gout, syphilis, diphtheria, and others, but, after having existed for a time, acquire an individuality of their own, and demand different remedial measures for their removal. Thus an eczema or a neuralgia may originate from a gouty condition of the blood, but may after a time become autonomous, and yield rather to local applications than to internal remedies intended to combat the gouty diathesis. In the same manner tabes, although owing to and first set up by syphilis, may, if it lasts for a number of years, become inaccessible to anti-syphilitic medication, and require a completely different treatment. I have never seen a patient more thoroughly syphilised than the one whose case is described on p. 264; yet he remained uninfluenced by anti-syphilitic treatment, and improved considerably under ergot, silver, and electricity.

The patient whose case is related on p. 176, recovered completely under the influence of the liquid extract of ergot, given in doses from half a drachm to a drachm three times a day for about eight months consecutively. It was a case in which there was no history or evidence of syphilitic infection. Ergot, like all other remedies, is occasionally disappointing, but is, nevertheless, a very valuable remedy for the affection which we are now considering, even where there is a history of infection.

Case 75.—In July, 1881, I was consulted by a merchant, aged forty-two, married, and father of three children, who had had syphilis ten years ago, and had for the last three years suffered from lightning pains, with ataxic gait, together with Romberg's and Westphal's symptoms. He was recommended to go to Aix-la-Chapelle, where he had two hundred inunctions without the least benefit. I now put him on ergot, under the influence of which he improved considerably. When he first came to me, he was so chilly that he had to sit in his office quite covered over with rugs; the bladder was so irritable that at night he had to get up at least four times to void the urine, and frequently wetted the bed. He was subject to bad attacks of lightning-pains, and his hands and feet were so benumbed that there was hardly any use in them. Six weeks afterwards his circulation was so much improved that he could sit for hours in his office without feeling chilly, and without rugs; the pain was much less severe; the bladder had quieted down to such an extent that he was only obliged to get up once in the night to pass his water, and he no longer wetted the bed. The sensation in his hands and feet had become normal, and he walked a great deal better.

On the other hand, Grasset[1] has lately seen ergot to do harm in the case of a male patient, who had suffered from tabes for some years, and who took at first four grains of the drug per diem, and had this gradually increased until he took fifteen grains per diem. On the second such day the patient appeared to have lost his voice and the power over all the limbs. Sensation was diminished; he had no pain, but he could neither sit up nor get up, and seemed completely paralysed. The ergot was then discontinued, with the result that these symptoms disappeared, and the patient gradually returned to his previous condition. This must be a very rare occurrence, as I have never seen anything even approaching to it. It shows, however, that the effects of the drug require to be carefully watched. Charcot is in the habit of giving ergot for four days, and then discontinues it for the next two, and so on.

Nitrate of silver was first recommended for tabes by Wunderlich in 1858, and has since then been largely used with varying results. At first observers were unanimous in their praise of the drug; but after a time it fell into disuse, and it is at present not so favourably looked upon as it was twenty-five years ago. I have all the time, since I first commenced the use of this preparation, remained faithful to it for certain cases, more especially where pain was a prominent symptom, and have often seen patients derive great advantage from it. I prescribe it chiefly in those cases in which there is no evidence of syphilitic taint, and where patients have had a sufficiency of anti-syphilitic treatment without being apparently much the better for it, as far as the cord-lesion is concerned. I generally give it in doses of one-eighth to one-fourth of a grain. It is important to recollect that all organic substances reduce nitrate of silver, whether in substance or in solution; and pills prescribed to be made up with liquorice, taraxacum, crumb of bread, etc., are, therefore, practically inert. This explains sufficiently why so many practitioners have seen no good results from the use of this drug; and it is singular to find so excellent a chemist as the late Peter Squire[1] directing it to be mixed up with crumb of bread, which contains sufficient chloride of sodium to decompose the silver completely. I have for years past been in the habit of prescribing the nitrate to be made up into a pill with argilla or bolus alba (silicate of alumina), which cannot possibly decompose it. The initial prescription, ordering the eighth part of a grain, reads as follows:—

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[1] "Progrès Medical," No. 11. Paris, 1883.

℞.	Argenti nitratis	gr. iii.
	Argillæ	gr. lx.

F. c. aq. dist. q. s. pilulæ no. xxiv., obduc. foliis argenti. S. a pill to be taken twice a day before meals.

More recently, Martindale[2] has recommended kaolin ointment as an excipient. Kaolin is native silicate of alumina, which has been purified from free silica and undecomposed felspar, and is made up into an ointment with equal parts of vaseline and paraffin.

Shortly before or after taking the silver pill, the patient must not take anything salty, as this would decompose the nitrate, and form insoluble chloride of silver. Our object must be to change the nitrate in the stomach into the albuminate of silver, which is soluble in diluted lactic and hydrochloric acids, and may, therefore, be absorbed; while chloride of silver, which is formed when table-salt is in the stomach, and also when somewhat large quantities of the nitrate are taken, is only soluble in ammonia, and evacuated with the fæces. It is, therefore, important that the patient should not partake of any highly salted food either before or after taking the pill; while a little milk taken immediately after it would tend to promote the formation of the soluble albuminate. The same considerations show that it is useless to prescribe large doses of the nitrate. Silver has been found in the urine and the bile, and, if given for a considerable time, will accumulate in different parts of the body.

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[1] "Companion to the British Pharmacopœia," p. 44. 8th edition, 1871.

[2] "The Extra Pharmacopœia," p. 50. London, 1883.

The slaty discoloration of the skin which is known as argyria, and caused by the prolonged exhibition of the nitrate, is fortunately now only rarely seen; and it must be pronounced unjustifiable to push the drug so far as to permanently disfigure the patient. Argyria when once established is incurable, the silver being deposited not in the epidermis, but in the corium. Removal of the epidermis by means of blisters has therefore not the least effect; nor does iodide of potassium or subsulphite of sodium, which have been recommended for internal use, eliminate the deposit. It is therefore our duty, when prescribing this preparation, to stop short of a quantity which would cause discoloration. Krahmer[1] has shown that the minimum quantity of the nitrate which will produce argyria is four hundred and fifty grains. The highest amount given should therefore be under three hundred grains.

According to Liouville, Ollivier, and Friedreich, albuminuria is apt to be induced by a prolonged administration of this drug. The urine should therefore be examined from time to time while the patient is under its influence. I must say that I have never found a trace of albumen in the urine under these circumstances; and its occurrence must therefore be exceptional.

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[1] Theodor Husemann, "Handbuch der gesammten Arzneimittellehre," vol. i., p. 464. 2nd edition. Berlin, 1883.

Tweedy,[1] of Dublin, has recorded the case of a patient affected with locomotor ataxy who had taken it persistently in doses of one-third of a grain for nearly twelve years. The symptoms of ataxy had completely disappeared, but the patient had become argyrised, and showed the peculiar leaden discoloration of the skin. On the other hand, Riemer [2] has seen a case of tabes where a patient consumed altogether 5,672 pills, and the first traces of argyria appeared after twelve months' use, with 2,900 pills, containing 1,740 grains of the nitrate, or 1,104 grains of metallic silver. This patient eventually died of phthisis, and there had been no improvement in the tabes! The peculiar discoloration was discovered after death not only in the skin, but also in the mucous and serous membranes, the blood-vessels, kidneys, mesenteric glands, and the connective tissue; the substance of the brain and spinal cord did not contain any deposit, but the choroid plexuses and the pia and arachnoid were full of silver, while the dura mater showed traces of it. The plexuses appeared as black as ink to the naked eye; it was difficult to dissociate them; the tissue had lost its elasticity, and the silver was found lying in dense black granules regularly arranged on the epithelium, where it formed a kind of silvery membrane.

Bokai,[3] of Pesth, has described a case of tabes in which the nitrate taken for three months, in doses varying from one-seventh to one-third of a grain per diem, cured the patient. Only the knee-jerk remained absent on both sides. Two years afterwards no relapse had occurred. Of course, such cases are exceptional; and it must be confessed that nitrate of silver often proves disappointing. Other preparations, such as the oxide, the phosphide (Hamilton), and the double salt of iodide of silver and potassium, seem to be even less certain in their action than the nitrate.

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[1] "Medical Times and Gazette," March 24th, 1883.

[2] "Archiv für Heilkunde," p. 296. Leipzig, 1875.

[3] "Orvosi Hetilap," No. 43. Buda, 1883.

In some cases I have found the subcutaneous injection of a silver salt, as recommended by Eulenburg, extremely useful. The phosphate has the disadvantage of not being readily soluble in water, while the hyposulphite is peculiarly suited for this administration. The formula for this is—

℞ Argenti chloridi recens præcipitati	gr. iii.
Sodii subsulphitis	gr. xviii.
Aquæ destill.	ʒ x.

Misce detur in vitreo fusco. ✓ S. For subcutaneous injection. Dose, from five to twenty minims.

The same precautions should be taken with this as with the injection of the perchloride of mercury (p. 302). Rosenthal prefers the acetate of silver for hypodermic injection. He directs three-quarters of a grain to be dissolved in two and a-half drachms of distilled water, and to inject of this ten minims. In two cases out of four thus treated, there was striking improvement.

Chloride of gold and potassium, which has been recommended by several observers, has proved completely inert in my hands. I have given it in doses varying from one-eighth to half a grain for several months consecutively.

One of the most important remedies which is applicable to all cases of tabes, whether of syphilitic or non-syphilitic origin, is *electricity*. Duchenne recommended long ago faradisation of the skin by means of a wire brush, in combination with iodide of potassium and mercury, and stated that he had seen good results from its use. This proceeding, which had for many years fallen into disuse, has recently again been strongly recommended by Rumpf,[1] of Bonn, who recommends to faradise the back, as well as the limbs, systematically for ten or twelve minutes each time. This proceeding appears to have occasionally been successful in his hands. The current-strength should be such as to cause contractions of the muscles supplied by the median nerve on applying the brush to the latter at the bend of the elbow. There is no question that the cord is reflexly affected by this proceeding, and it is probably owing to this that any therapeutical results are obtained. I prefer faradisation of the skin as an aid to the constant current, rather than as a substitute for it.

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[1] "Aerztliches Vereinsblatt," No. 10, 1881; "Neurologisches Centralblatt," Nos. 1 and 2, 1882.

Faradisation of the skin in tabes requires great familiarity with the practice of medical electricity, if it is to do good. An excessive dose may do great harm; and the operator must be well acquainted with the different degrees of susceptibility of the skin in different parts of the body to faradism. The wire brush used should have a large surface, not less than the size of half-a-crown, and must be quite soft. The small prickly brushes usually sent out with induction-machines are worse than useless. Messrs. Coxeter and Son have made a very nice wire brush for me, which can be strongly recommended. The current-strength used must always be moderate, and proportionate to the state of sensibility. Where anæsthesia is present, more force should be used than where sensation is not much impaired. I have at present a patient under my care for whom I use on the left side six, and on the right side eighteen, degrees of Stöhrer's induction-machine.

As far as electricity is concerned, however, we must look to the constant current as our sheet-anchor in these cases. The constant current is not able to restore central nerve-tubes which have been destroyed; but it has the tendency, by its catalytic and stimulating effects, to improve the nutrition of those which are beginning to be diseased, to strengthen the remainder, and also to some extent to call into play the law of substitution, thus causing healthy fibres in neighbouring strands to perform the work of others which have perished.

The merit of having introduced the constant current into the therapeutics of tabes belongs to Remak, sen., whose publications were, however, more those of the enthusiast than of the sober observer (1858). Subsequently this treatment was largely resorted to by Benedict of Vienna, Moritz Meyer of Berlin, and myself, with the result that the remedial value of this form of electricity in tabes was placed beyond dispute. More recently Erb and his pupils have added their testimony to ours, and at present there are few cases of tabes which are not subjected to this mode of treatment. Insufficient acquaintance with the technicalities of the electric treatment has, however, frequently been a source of disappointment. It is not desirable that the novice in electro-therapeutics should win his first spurs in cases of tabes. Patients of this kind are generally extremely sensitive, and errors in the mode of application are apt to be punished by a speedy deterioration of their condition.

The principal rule in applying the current is, that the applications should be short and gentle. I frequently use only one or two milliampères, and consider Löwenfeld's[1] recommendation of five to fifteen milliampères risky. We must, however, act according to individual circumstances; for where the principal symptoms are anæsthesia and loss of power, a greater current-strength may be used than where we have to do with lightning-pains, paræsthesia, and tendency to crises of different kinds. The application of large electrodes (about five inches by two) to the spine is the most important proceeding. Here, again, the aspect of the individual case has to guide us to a considerable extent. Where signs of irritation preponderate, the cathode should be placed at a distance from the spine. In such cases I put the large anode to the region of the lumbar enlargement, and the cathode to the epigastrium, for two, three or four minutes at a time ; afterwards I place the anode to the cervical spine, and the cathode on the sternum, for from two to four minutes. Where debility without irritation seems prominent, the cathode is placed to the cervical spine, and the anode immediately below it ; the latter is left *in situ* for a minute, or a little more, then moved further down, left on again, and so until the whole of the spine has received its influence, the whole application lasting about five minutes.

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[1] "Ueber den gegenwärtigen Stand der Therapie der chronischen Rückenmarkskrankheiten," p. 20. München, 1884.

Moritz Meyer and Erb prefer the application of the cathode to the region of the cervical sympathetic nerve, the anode being placed to the opposite side of the spine, and gradually conducted downwards. This is a useful application, but is frequently not well borne, as shown by giddiness, swimming in the head, buzzing in the ears, and other unpleasant symptoms. I therefore prefer one of the direct applications to the spine which I have mentioned, with a current of moderate force, and then add a still more gentle application to the sphere of the cervical sympathetic nerve, for one or two minutes at a time at each side. For the latter I have occasionally used only the fifth or tenth of a milliampère, and yet with decided results.

Neftel recommends an application to the head as well as to the spine. For the latter he places the cathode to the neck, and the anode to the lumbar spine ; he begins with a feeble current, which is then gradually increased. After three minutes the current-strength is again diminished, and the anode is then passed several times slowly down the spine, over the spinous as well as the transverse processes. Meyer and Brenner speak highly of the application of the current to tender points, which may be discovered about the spine. Such tender points are, however, only rarely met with, and it is still doubtful whether much importance is to be attached to them.

Certain local applications of the current may be also used with advantage, in order to stimulate the action of the bladder and the bowels, to relieve pain and anæsthesia, etc. These, however, should only play a secondary part in the treatment, the central applications being always the most important.

Galvanic or faradic baths, as at present used, constitute a form of quackery which should be strongly discountenanced in the treatment of tabes.

Another means, of which some observers have seen considerable results, is *water* in the widest sense of the word. Patients with tabes are frequently treated in hydropathic establishments, and the peculiar fervour which is characteristic of most water-doctors has been shown in their reports of the success achieved in such cases. It has, however, gradually been ascertained that the various applications of hot as well as cold water do harm in place of good. Sea-baths, vapour and Turkish baths, ordinary cold baths, the douche in its various forms, and those mineral baths the chief feature of which is their heat, often cause a rapid deterioration in the state of the patient. On the other hand, it has been found that tepid baths, of a temperature varying from 80° to 90°, and of short duration, that is to say, from four to ten minutes, act beneficially; and this principle holds good not only for ordinary water, but also for mineral waters.

Rosenthal[1] states that a judicious hydrotherapeutic treatment allays central irritation, strengthens the nervous system, and diminishes its undue excitability and the dangers to which it is exposed on account of its sensibility to atmospheric changes. He recommends chiefly friction with a cloth dipped in water between 55° and 65°; a cold compress should be applied to the head, and the patient be placed in a bath of 75° to 68°, into which cold water is slowly poured until the temperature is lowered to 64° or

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[1] "Diseases of the Nervous System," vol. i., p. 259. (American edition.) New York, 1879.

61°. The patient should remain in this bath from four to eight minutes ; he is then showered, and friction applied to the back. When the procedure is over, the patient should feel thoroughly comfortable, and be enjoined to take some exercise in the open air.

Mineral waters and baths have been employed for the cure of tabes ever since the disease has been known, and generally with unfavourable results. The thermal springs of Gastein, Wildbad, Teplitz, and other similar Spas, which are so useful for many forms of rheumatism, gout, neurasthenia, and other conditions, are utterly unsuitable for tabes. The journey to and from a Spa alone often does a great deal of harm. In recent years the brines of Oeynhausen and Nauheim have acquired considerable reputation. I regret to say that I am unable to speak well of their effects. All the patients whom I have sent to Oeynhausen returned in a worse condition than they left : one had a stroke of paralysis while there; and in another, soon after his return home, an acute form of general paralysis of the insane supervened, rendering it necessary that the patient should be placed under restraint.

The Aix-la-Chapelle treatment, which under the auspices of Drs. Brandes, Reumont, Schumacher II., and others, has become an established institution, consists of the simultaneous use of mercurial inunction and the external and internal use of the sulphurous waters of that Spa. It is believed that by the use of these different agents at the same time, more benefit is produced than by either of them singly; and there can be no doubt, from the positive statements of reliable observers, that the Aix-la-Chapelle treatment is frequently successful in tabes. It is, perhaps, in the nature of things that I should only have seen patients in whom it has been either useless or appeared to do harm ; yet I have found this so frequently to be the case that I feel it incumbent upon me to reserve my judgment about the real merits of the Aix-la-Chapelle treatment in tabes.

French observers express the highest opinion of the mineral waters of La Malou, near Montpellier, in the department of l'Hérault, which contain iron, alkalies, and traces of arsenic, with carbonic acid gas, besides which they have a high temperature. Grasset[1] quotes some wonderful cures from a little book of Privat's, which are worth mentioning. A man, aged thirty-seven, fell into the water in winter, and was obliged to keep his wet clothes on for eight hours subsequently. Five weeks afterwards lightning-pains, constipation, sluggishness of the bladder, and sexual debility supervened. Fifteen months afterwards there was right ptosis and double vision, ataxy, incontinence of the urine, impotency, and anæsthesia in the lower limbs. In this state the patient arrived at La Malou, walking with crutches. He took twenty baths, after which the pains disappeared, and he was enabled to return to work. Three years afterwards, however, he had a relapse owing to exposure, and the disease after that ran its usual course. More extraordinary is the case of a doctor who was, at twenty-nine years of age, affected with erratic pains, motor incoordination, strabismus, paralysis of the third nerve, gastralgia, dyspepsia, obstinate constipation, sluggishness of the bladder, absolute impotency, and plantar anæsthesia. This went on for eight years, the last two of which he spent in his room or bed. Two successive seasons at La Malou were followed by considerable improvement. The year after he did not return there, and lost what he had gained. He then came back, and had two courses of treatment annually, for four or five years consecutively. The pain gradually diminished; the motor power was fully re-established; and ultimately the plantar anæsthesia disappeared nineteen years after the beginning of the malady. Tabes in La Malou seems occasionally to assume extraordinary forms; thus Privat mentions the case of a man who could only walk with the aid of a stick or an arm, but who, during his crises of somnambulism, which came on every eight or ten days, could walk quite easily and without a stick! Seeing the extraordinary benefit which patients seem to derive at La Malou, it is singular to find Grasset stating, almost on the same page, that "the prognosis of tabes is most serious, and that it is an incurable disease"! The description of the cases just given is not sufficiently detailed to enable me to speak definitely about their nature; but as Charcot and Combal, of Montpellier, appear to send patients with tabes habitually to La Malou, it is possible that the treatment there may occasionally do good. We must, however, be cautious in accepting the statements of local Spa doctors, who are invariably inclined to take a rose-coloured view of the effects of their own waters.

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[1] "Traité Pratique des Maladies du Système Nerveux," p. 338. 2nd Edition. Montpellier, 1881.

Nerve-stretching, for the relief or cure of tabes, has had a short and not very brilliant career. The operation was originally introduced into surgical practice by Nussbaum, of Munich, in 1872, for the relief of pain, and proved successful in an intractable case of "painful spasm in the left arm." The first English surgeon who performed the operation was the late Mr. Callender, who cured a neuralgia in an amputation-stump by stretching the median nerve (1875). Since then nerve-stretching has been frequently resorted to, in the belief that the effects of it were peripheral, but more decided than those of neurotomy or neurectomy. It was assumed that the molecular condition of the nerve was altered; that undue excitability in the motor as well as in the sensory sphere was diminished; and the success of the proceeding was ascribed to the breaking up of adhesions of the nerve with the surrounding tissues. Vogt[1] of Greifswald, whose able essay on this subject appeared in 1877, came to the conclusion that, by stretching, the nerve-fibres were separated from the neurilemma, that the blood-vessels in the sheath of the nerve were stretched and loosened, and that thereby the nutrition of the nerve itself was improved. He denied that the operation had any influence on the nervous centres.

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[1] "Die Nervendehnung in der chirurgischen Praxis." Leipzig, 1877.

In 1879, the medical world was startled by a publication of Langenbuch,[1] of Berlin, in which nerve-stretching was strongly recommended for the cure of tabes. A patient, who was believed to be suffering from that disease, was subjected to stretching of the sciatic and crural nerves, apparently with benefit as far as pain and ataxy were concerned. Three months afterwards, the pain being severe in the arms, the brachial plexus was stretched by the same surgeon, with the result that the patient was seized by an epileptiform convulsion, and died on the operating-table. The spinal cord was sent to Professor Westphal for examination, and he found that the case had not been one of tabes, nor indeed of any spinal disease.

Although, therefore, the first step in this matter was hardly encouraging, Langenbuch nevertheless continued to operate on patients suffering from tabes, and started the theory that this operation had a beneficial influence on the nervous centres. He stated that, by stretching, the morbid products of sclerosis were necrosed, and therefore rendered fit for absorption, while the operation at the same time afforded a powerful stimulus to the remainder of healthy nerve-tubes, and thus enabled them to do their work better. Carried away by his enthusiasm for this procedure, the same surgeon actually stretched the nerves of the sphincter ani for tenesmus of the bowel, and the pudendal nerves in order to stop the habit of masturbation!! The latter operation was followed by septicæmia, convulsions, and death.

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[1] "Über Dehnung grosser Nervenstämme bei Tabes Dorsalis." "Berliner klin. Wochenschrift," No. 49, 1879; also Nos. 24 and 27, 1881; also Nos. 12 and 13, 1882. Langen*buch* has, more especially in this country, been frequently confounded with the veteran surgical master, Von Langen*beck*; and on account of the similarity of the two names, particular attention has been directed to this matter.

Langenbuch's publications naturally aroused much attention in the city where he performed his operations ; and the results which he asserted had been obtained were subjected to a somewhat searching criticism, which proved decidedly unfavourable. Thus he had reported a case as greatly improved, which had been seen by Remak, jun., both before and after the operation ; and Remak reported that the patient was worse after the stretching. In another case of quite recent tabes, Bernhardt reported that, six weeks after the operation, the right leg, in which the crural nerve had been stretched, was paralysed ; that the patient then suffered from severe intercostal neuralgia, and some time afterwards had lightning-pains in the legs. Before the operation the patient could walk pretty well, but five months afterwards his legs were quite useless. In a third case, in which Langenbuch had reported improvement in cutaneous and muscular sensibility, in walking, urination, the function of the sexual organs, etc., Bernhardt found the patient in exactly the same condition in which he had been before the operation.

Nerve-stretching is by no means devoid of risk.[1] A somewhat considerable number of fatal cases has been recorded by Socin, Billroth, Berger, Benedict, Riegner, Hahn, Müller, Gussenbauer, Hirschfelder, Fluger, and others. In some of these, undue violence in stretching appears to have been the cause of death, the medulla oblongata having apparently received a shock at the time of the operation. In one case, severe vomiting and singultus, together with complete paralysis of the bladder and bowels, supervened after the operation ; dyspnœa and cyanosis eventually set in, and the patient died comatose on the ninth day. In other cases the patients have died of blood-poisoning from septicæmia, owing chiefly to infection of the wound by the urine and fæces, etc.

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[1] Vide my note on "The Dangers of Nerve-stretching," "British Medical Journal," Jan. 7, 1882.

The literature on nerve-stretching is already so enormous that it is utterly impossible, in the limits of the present treatise, to even allude to all the experiments which have been made on this subject ; and we must refer those more particularly interested in it to the admirable lecture recently delivered by John Marshall before the College of Surgeons, and to the able and pains-taking treatise of Stintzing[1] of Munich. The latter concludes from his experiments on animals that stretching a healthy mixed nerve has in general a paralysing effect on the same, which extends pretty equally to the motor, sensory, vaso-motor, and trophic fibres. The degree of the paralysis is proportional to the force used in stretching, and the symptoms correspond on the whole to those of degenerative atrophy of the nerves, although they show numerous deviations from the typical course of the latter. Even where the paralysis has progressed very far, and where the force used in stretching has been more than half the body-weight of the animal, repair to a considerable extent may take place.

These results are certainly very far from encouraging, and do not afford any explanation of the slight therapeutical effects which have unquestionably in a few cases been obtained. Stintzing has given a careful description of four cases of tabes, in which Nussbaum stretched the sciatic nerve, either by cutting down upon it or subcutaneously, and has come to the following conclusions[2] :—

"Nerve-stretching has an influence on tabes, which is due to its action on the cord itself, as shown by the effects on other nerves which have no connection with the one that has been stretched. Remote effects of this kind take place transversely, as well as longitudinally, throughout the cord. They are partly stimulating, partly paralysing, and are shown in the sphere of motion, co-ordination, the function of the bladder and rectum, the secretory functions, the sensory and reflex actions. Sensibility is affected in all its different forms, viz., contact, pressure, farado-cutaneous, temperature, and pain. The most constant result is the relief of pain, and it is for this that the operation should be chiefly performed when other means have failed, more especially as subcutaneous stretching of the sciatic nerve is entirely devoid of risk. Should this latter operation, however, fail, the cutting operation is not allowable where, in consequence of loss of control over the bladder and bowels, infection of the wound may be feared."

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[1] "Über Nervendehnung. Eine experimentelle und klinische Studie." Leipzig, 1883.

[2] *Loc. cit.*, p. 166.

Stintzing's statement, that subcutaneous stretching of the sciatic nerve is entirely devoid of risk, must, however, not be taken literally; for Baum has recorded the case of a young man who died of collapse during that operation. Inspection showed multiple hæmorrhages along the sciatic nerve, in all the intervertebral foramina, and in the dura and pia mater up to the cervical portion of the cord. I have, therefore, arrived at the opinion *that nerve-stretching is, under any circumstances, a hazardous operation in tabes;* and I have recently ceased to recommend it.

A few words must be said about some other remedies which should *not* be used in this disease. Of these, the principal one is strychnia, which I have known to do harm, whether given *per os* or subcutaneously. Counter-irritation of the spine was condemned by Romberg more than forty years ago, when hardly ever a patient came to consult him whose back did not show numerous cicatrices owing to the seton, the actual and potential cautery, the moxa, and cupping. While in Pott's disease, and in chronic pachymeningitis counter-irritation to the spine is justifiable, and not unfrequently followed by tolerably good results, the same treatment applied to tabes can only injure by the suffering it causes. As a matter of fact, the only form of counter-irritation which is now used in tabes, more especially in France, is the "pointes de feu" by means of Paquelin's gas-cautery; and those who use the latter profess to do so, not for the purpose of curing the disease, but for the relief of pain.

For the latter we have a number of other remedies which often prove useful. Amongst them are faradisation of the skin; application of a very small circular anode conveying the constant current, the large cathode being placed at a distance from the affected part; subcutaneous injection of plain water (p. 154), and of morphia and atropia, followed by a few whiffs of ether; local applications of a belladonna and chloroform liniment, of aromatic spirit of ammonia, and of ether-spray; and for internal use the salicylate of soda in ten or twenty grain doses; hydrate of chloral; tincture of gelseminum; and bromide of uranium, in pills containing one-seventieth part of a grain.

For optic atrophy, De Wecker has recommended stretching of the optic nerve! and Kümmell, of Hamburg, has actually performed this operation in three cases, in one of which the results are stated to have been good, while in the other two they were negative. Galezowski has recently recommended subcutaneous injections of cyanide of gold, one-fourteenth grain as a dose; while subcutaneous injection of strychnia has been practised for years past without producing much, if any, effect. As I have found the general effects of strychnia injurious in tabes, I would strongly dissuade practitioners from resorting to this drug whether for optic atrophy or other symptoms.

Where nocturnal emissions of semen are troublesome, bromide of ammonium, combined with hydrate of chloral and tincture of lupulus, proves useful. For atony of the bowels I can strongly recommend the fluid extract of Cascara Sagrada, given in doses of from ten minims to a fluid drachm, once or twice a day. For nocturnal incontinence of the urine a piece of goldbeater's skin may be placed over the opening of the urethra and fixed with collodion ; and in catarrh of the bladder, with ammoniacal decomposition of the urine, salicylate of soda internally, and washing out the bladder with antiseptics, are useful.

CHAPTER X.

FRIEDREICH'S DISEASE.

THE pathology and causes of this form of sclerosis have already been discussed (pp. 65 and 112). We have seen that it constitutes a diffuse form of sclerosis of different portions of the spinal cord and medulla oblongata, which is clinically as well as anatomically distinct from tabes and from insular sclerosis; and that it tends to appear in several members of the same family about the time of the development of puberty, and in some cases even at an earlier age.

The malady generally commences without any apparent exciting cause or premonitory symptoms, with a feeling of muscular debility in one or both legs. Walking becomes difficult, but does not show the peculiar features of Duchenne's ataxy. The gait is staggering, like that of a drunken person, but the want of co-ordination is not increased by shutting the eyes. After a time the trouble spreads to the upper extremities, and the finer movements of the hands and fingers become impossible. The affection assumes occasionally the hemiplegic form. The head is sometimes seen to oscillate like that of a person who goes to sleep on a chair; and this tremor is increased when the patient voluntarily moves his head.

There is also a peculiar form of nystagmus, showing want of synergy in the muscles of the eyeballs. Friedreich[1] and Seeligmüller[2] have found this to be one of the later symptoms of the disease, and distinguished from ordinary nystagmus—such as is seen in children together with opacities of the cornea and lens, or strabismus, in albinos, etc.—by its non-occurrence during rest. In the usual form of nystagmus there is at all times a peculiar restless oscillation of the eyeballs, generally in a horizontal direction, but occasionally rotatory; while the nystagmus of Friedreich's disease only appears when the patient attempts to fix his eyes on an object; and its direction is then either horizontal, vertical, or diagonal. If an object be moved from one part of the visual field to another, and the patient be told to follow it with his eyes, jerky motions of the eyeballs are perceived, which clearly show ataxy or asynergy, being irregular in character and much slower than those of ordinary nystagmus. Friedreich considers this nystagmus to be owing to disease of the nuclei of the ocular muscles on the floor of the fourth ventricle.

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[1] "Archiv für Psychiatrie," vol. vii., p. 235. 1876.

[2] *Ibid.*, vol. x., p. 222. 1879.

After a time the speech becomes slow and drawling, and eventually quite unintelligible. The tongue, however, appears to be freely movable in all directions, can be easily protruded, and held out without any appearance of tremor; yet in the later stages of the disease there may be tremor in the tongue and glossoplegia. Towards the end we observe more or less complete muscular paralysis and atrophy, and occasionally pain, cramps, and rigidity.

A curious feature of the complaint is, that sensibility does not suffer until the very last; while in ordinary tabes lightning-pains, areas of hyperæsthesia and anæsthesia, numbness in the soles of the feet and the sphere of the ulnar nerve, are early symptoms. The sphincters do not suffer, and there is for a long time no tendency to bed-sores. The cutaneous reflex sensibility and the electric tests are normal. The tendon reflexes have not been studied in the earlier cases, but, where they were investigated, were found to be absent. The intellect is normal, and the special senses do not suffer. Argyll-Robertson's symptom, which is so common in ordinary tabes, is absent in the complaint now under consideration. In men there is impotency; and in women menstruation is irregular and unsatisfactory. Less constant symptoms are curvature of the spine and a peculiar form of vertigo, which comes on in paroxysms, and is not influenced by the position of the patient when the attack begins.

The course of the malady is exceedingly slow; and it is only quite towards the end that sensibility becomes affected, and that there is tendency to sacral bed-sores and catarrh of the bladder, with pain and cramp in the muscles. Symptoms pointing to an affection of the medulla oblongata are then apt to make their appearance, viz., acceleration of the pulse, excessive perspiration and salivation, and diabetes insipidus. Death is preceded by Cheyne-Stokes's respiration. In one case the affection lasted altogether thirty-one years; in another twenty-six; and where death took place at an earlier period, it was owing to typhoid fever, to which these patients seem specially apt to succumb. They appear to possess only slight powers of resistance to the typhoid poison, and collapse is apt to set in at an early period of the malady. This was in some cases no doubt promoted by an excessive fatty degeneration of the heart, which was found after death.

From what we have said it will be apparent that the disease is as different from ordinary tabes as one form of Bright's disease from another; indeed, the symptoms resemble rather more those of insular or multiple cerebro-spinal sclerosis than the common form of tabes. Sclerosis in patches is also liable to become developed towards puberty; there is no sentient or sensory trouble; the sphincters act well, but there is an affection of speech and vertigo. On the other hand, the peculiar form of tremor which is seen in multiple sclerosis does not occur in Friedreich's disease; and there are exaggerated tendon-reflexes, spastic gait, and muscular rigidity, strabismus, diplopia, and impaired intellect, in multiple sclerosis.

The diagnosis of Friedreich's disease can therefore rarely present any difficulties. Its prognosis is gloomy, and no kind of treatment has as yet appeared to be able to arrest the progress of the malady, much less to improve or cure it.

CHAPTER XI.

SPASTIC SPINAL PARALYSIS.

THE pathology of *spastic spinal paralysis*, or *spasmodic tabes dorsalis* has been shown (p. 51) to be as yet in a most unsatisfactory condition, and to require a great deal of further elucidation by careful anatomical investigations. Although there is *primâ facie* evidence to connect this disease with primary sclerosis of the lateral columns, and more especially of that portion of it which is known as the crossed pyramidal strands (p. 11), yet it cannot be said that this has been convincingly proved, and we shall, therefore, be on safe ground in reserving our opinion on the actual anatomical base of this malady.

The causes of the complaint are somewhat better known. We have seen that eating bread mixed with the meal of lathyrus cicera will give rise to it (p. 71), not only in men, but also in horses (p. 73); that the influence of the neurotic constitution is much more marked in its production than in that of tabes; that sex and age appear to have very little influence; and that a syphilitic history is much more rarely obtained in this affection than in tabes (p. 123).

The chief symptoms of spastic paralysis appertain to the motor sphere, and consist of loss of power in the limbs, rigidity and spasm of the muscles, and exaggeration of the tendon reflexes. The paresis or paralysis generally affects at first either one or both lower extremities, and the patient's first complaint is, therefore, of a difficulty in walking. He soon gets tired, and is apt to drag one or both legs on the ground. Some patients feel the greatest difficulty on rising in the morning, and improve as the day advances. The legs feel heavy, stiff, and weak ; the loss of power gradually becomes more marked, and eventually merges into paresis and complete paralysis. At no time, however, is there any ataxy of movements, and it makes no difference to the patient whether he stands or walks with his eyes closed or open.

I have found it most useful, in my examination of these cases, to measure the exact degree of muscular force which may be present, with a dynamometer constructed for me by Weiss and Son, for ascertaining the condition of muscular power in the lower extremities. The patient puts his foot on this instrument, first in the sitting position, and presses as hard as he can ; the excursion of the index is then noted, and the latter replaced ; he then gets up and puts the same foot on the instrument, resting the entire weight of his body on it ; the excursion is noted again, and the proceeding repeated for the other side. I find the healthy averages to be as follows:—

Right side, sitting	140°
„ „ standing	160°
Left side, sitting	130°
„ „ standing	140°

Now in spastic paralysis a considerable diminution of these numbers is obtained, according to the degree of loss of power which may be present. If these numbers are entered each time the patient is thus examined, an objective record of improvement or deterioration will be furnished. In somewhat advanced cases the index remains at zero.

The use of this instrument is also valuable for diagnostic purposes. My colleague, Hughes Bennett,[1] has drawn attention to the circumstance, which has also been dwelt upon by Müller, of Gratz, that young women may exhibit all the signs of primary spastic paralysis, simulating sclerosis, and yet eventually recover. In some such cases there are indications that there is a general hysterical condition, but in others all signs of hysteria are wanting. Bennett laments that there is not a single point by means of which we are enabled to differentiate these two conditions, although this is undoubtedly of great practical importance; for many unfortunate women suffering from spinal disease are considered hysterical, while others, who by a vigorous régime might be rapidly cured, are incarcerated for life as hopeless invalids. It is commonly assumed that nothing is easier than to distinguish a functional from an organic lesion, while in reality, in some cases, nothing is more difficult. This is evidenced by the fact that in some instances sclerosis and pseudo-sclerosis have only been distinguished on the post-mortem table, by the most distinguished physicians. It is therefore important to know, that the dynamometer which I have had constructed for measuring the force in the lower extremities, will, at least in a certain number of cases, enable us to distinguish between the functional and the structural form of spastic paralysis. In the former, although the patient may be unable to walk, the dynamometer often indicates a considerable degree of muscular power; while in the latter, more especially where the disease is somewhat advanced, the index of the instrument will only indicate 20° or 30°, in place of 140° or 160°, and occasionally will make no excursion at all.

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[1] "Medical Times and Gazette," Nov. 3, 1883.

Symptoms of motor irritation are superadded to the loss of power at an early period of the complaint. The legs are occasionally jerked about suddenly, especially after exertion and in bed; or there is a peculiar kind of tremor, or trepidation, which goes on for a considerable time, begins generally in the feet, and spreads from there to the legs, thighs, and body. If the patient be walking or standing at the time when this tremor commences, he occasionally stamps the ground with such force as to shake the room in which he is, and the noise may be heard at a considerable distance. Such symptoms appear to occur spontaneously, but are, in reality, owing to reflex irritation ; for they may be caused at any time by a sudden attempt to bend the knee, or by dorsal flexion of the foot. As soon as an attempt at voluntary movement, more especially of the body, pulls and stretches the tendons ever so little, this kind of tremor is produced ; and it may be arrested by plantar flexion of the foot, whereby the mechanical irritation is counteracted.

At the same time a peculiar stiffness and rigidity of the muscular substance is noticed. This is at first quite temporary, and chiefly noticed in walking, or when the legs are examined and passive movements are attempted ; but after a time it becomes more permanent ; and eventually complete and permanent contractions, more especially in extension, are produced. When the patient is in bed, the legs are held in extension and adduction, and great difficulty is experienced in changing their position. The adduction of the legs may be so strong that the knees can hardly be separated ; at the same time the hip-joints are slightly flexed, and the legs everted. The rigidity is owing to reflectory tension of the muscles, and is, more especially in the beginning, increased by the weight of the legs as they stand on the ground, or by the least active or passive movements which irritate the tendons. When the legs are well supported, so that any irritation of tendons is avoided, the rigidity is lessened.

In connexion with this point it is important to know that the muscular stiffness may be greatly diminished by placing the patient in a warm bath. The legs are then carried by the water, and lose a portion of their weight, so that the pulling and stretching of tendons which is simply owing to weight is more or less avoided. The legs are then found to be no longer fixed in extension, and passive movements are comparatively easy. Percussion of tendons has, however, the same influence in the water as in the air. It is only the irritation of tendons caused by weight and pulling which is lessened, and indeed remains lessened for some time after the bath. Prolonged warm baths are therefore very useful in the treatment of this affection.

The combination of loss of power and rigidity leads to a peculiar kind of a walk which has been first described by Erb as the "spastic gait."[1] The feet seem to be tied to the ground, which they scrape; and any slight unevenness, which they may happen to encounter on it, becomes a great impediment to progress. When the patient goes downhill, he appears to be dragged along by the simple weight of his body; he is obliged to hurry, and is in danger of falling with his face forwards. He walks habitually on tiptoe, with the heels lifted up, by contraction of the gastrocnemii; and the points of the boots are therefore worn off sooner than the soles or the heels. The whole body is generally lifted with each step, and may be either thrown backwards or forwards. The legs are too much adducted, and can only be separated by considerable efforts on the part of the pelvic muscles; and it may be easily imagined that a walk with such difficulties is exhausting to the last degree.

It is singular that patients of this kind, when they are hardly able to crawl along, can stand without an effort for almost any length of time. A gentleman who was sent to me in November, 1871, by Mr. Maclaren, and in whom I have had the opportunity of watching the gradual evolution of this disease during the last thirteen years, related to me some curious instances of this. On one occasion when his walking was already very bad, he went to the Derby and had the greatest difficulty to walk from his carriage to the stand with the help of two friends; but, once there, he stood for five hours consecutively and watched all the races without any fatigue. The next few days he thought that he walked a great deal better. On another occasion, in 1872, he stood, on the Thanksgiving Day for the recovery of the Prince of Wales, in an open shop for five hours consecutively, watching the procession, and again walked much better the next few days. It then occurred to him to utilise standing for the treatment of his complaint, and he went shortly afterwards to the Gaiety Theatre, and stood out a whole performance there. This, however, did not succeed; for he felt much worse afterwards, which he explained by the air in the theatre having been very hot and stuffy.

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[1] "Krankheiten des Rückenmarks," vol. i., p. 96. Leipzig, 1878.

After a time which may extend over several years, the upper extremities are likewise affected. Loss of power is experienced in the hands; the patient has a difficulty in taking hold of objects, in writing, in carving, in dressing himself, and more especially in buttoning his clothes. He finds it difficult to lift the arm, and to bend the elbow. Stiffness and rigidity of the muscles is likewise not long in making its appearance, and affects then chiefly the flexors and adductors. The fingers are more or less clenched into the palm of the hand, the wrist is pronated and flexed, the elbow semi-flexed or extended, and the arms are adducted to the sides of the body. Occasionally, however, I have seen, even at an advanced stage of the affection, pure paralysis without rigidity in the muscles of the upper extremities.

The disease soon afterwards invades the sacro-lumbar and abdominal muscles, causing loss of power and rigidity in them. The abdomen is hard and prominent, and separated from the base of the thorax by a deep horizontal ridge. Respiration is then apt to become difficult, although at this time there may be no affection of the medulla oblongata. The patient can no longer sit up in bed, and gradually becomes more thoroughly helpless.

Occasionally the disease assumes the form of hemiplegia or cervical paraplegia, so that first one leg and then the arm of the same side is affected; or both arms are first invaded, and the lower extremities follow suit some years afterwards.

The next symptom of importance which we have to consider is the exaggeration of the *deep reflexes*, which contitutes one of the most characteristic features of the disease. The knee-jerk is more particularly exaggerated, so that the slightest touch of the ligamentum patellæ with the finger or percussion hammer causes the leg to fly forwards. Another mode of eliciting it is to fix the patella and push it down suddenly, when clonic convulsions may be produced. Percussion of the substance of the quadriceps will have the same effect, the degree of the response being inversely proportionate to the distance of the point percussed from the patella. It is, however, not only the tendon reflexes, but also the periosteal reflexes which are increased; for percussion of the tibia, more especially its internal edge, may likewise cause the knee-jerk to appear. This is an interesting circumstance, as it speaks very strongly for the reflectory and against the mechanical theory of these phenomena. Indeed it seems impossible to assume that the knee-jerk thus produced could be owing to a propagation of a mechanical concussion to the muscle, inasmuch as the slightest blow struck on the lower end of the tibia, which could not have any effect on the thigh, will elicit the response, while powerful blows on other parts of the leg have no influence whatever in causing any contraction of the quadriceps.

Ankle-clonus, which is also called "foot-phenomenon" or induced trepidation, is another important reflex, and must be looked upon as an exaggeration of the normal reflex of the tendon Achillis. It is produced by suddenly lifting the toes or, better still, the front part of the foot with one hand, while the patient's leg is supported under the knee-joint with the other hand. We then observe a series of rhythmic muscular movements or oscillations of the affected leg, which go on for a time varying from half a minute to five minutes and more. In advanced cases the irritation crosses over to the other side, so that both legs are shaken by clonic convulsions, and the phenomenon is then called "spinal epilepsy"—a most unsuitable term which should be discontinued. Brown-Séquard has stated that ankle-clonus may be at once arrested by passive plantar flexion of the big toe; but such is not the case, as it is only arrested if plantar flexion of the *foot* is made. This latter proceeding neutralises the mechanical irritation of the tendon Achillis, and therefore arrests its consequences. The big toe may be ever so much flexed, and yet the ankle-clonus will continue, provided plantar flexion of the foot is avoided. On the other hand we find that, where the gastrocnemii are much contracted, the production of ankle-clonus may be rendered difficult or impossible, as a certain amount of relaxation of the tendon is essential for the causation of the phenomenon. In some cases percussion of the tendon Achillis will be found more effective in producing the clonus than dorsal flexion of the foot.

Further tendon-reflexes which may be elicited in an exaggerated form in the lower extremities in spastic paralysis are those of the glutæi and the adductors of the thighs, produced by percussion of the lumbar spine; and of the biceps femoris, the semi-tendinosus and semi-membranosus, and the anterior and posterior tibiales, by percussing their respective tendons.

In the upper extremities, likewise, a great variety of exaggerated deep reflexes may be produced. By percussing the vertebræ of the cervical spine, various muscles of the arm may be caused to contract; by tapping the collar-bone, the pectoralis major and the biceps may be affected; and by percussion of the lower end of the radius and ulna, and of the bones of the carpus and metacarpus, reflexes in neighbouring and distant muscles may be elicited. Crossed periosteal reflexes are also seen, so that on percussing the collar-bone of one side a contraction occurs in the biceps of the other side. Percussion of the internal condyle of the tibia will cause the adductors of the thigh to contract powerfully on the same side, and rather less strongly on the opposite side; and a tap on the sternal ends of the upper ribs may cause contraction of the opposite pectoralis major.

In some cases considerable differences are noticed in the degree of exaggeration of the deep reflexes on the two sides, more especially with the patellar reflex; and it is then generally found that such exaggeration is proportionate to the loss of power in the limb. In a patient whom I saw with Dr. Mackintosh, of the Brompton Road, in April, 1882, and in whom the right leg was worse than the left, the knee-jerk was much more violent in the right than in the left side, and ankle-clonus could only be produced in the right, but not in the left foot.

If a muscle is made to contract by percussing its tendon, the same may be brought about in a general way by directly percussing its substance. A knowledge of the motor points of the muscles, which is so important in carrying out faradisation, is useful, as it is chiefly from these points that such contractions may be elicited. Strümpell[1] contends that such a contraction is frequently a reflex proceeding from the fascia of the muscle, and may then affect the entire belly of it just as if the tendon had been percussed. This may be seen in the gastrocnemius, but more especially in the semi-membranosus and semi-tendinosus muscles; and the fact that the effect is greater by percussing the upper than the lower portions of the muscle—that is, farther away from the tendon—appears to speak against the phenomenon being owing to mechanical transmission of the concussion to the tendon. This, however, does not hold good for all cases; for I have already remarked that in spastic paralysis the contractions produced by percussion of the quadriceps are generally all the more marked the nearer the blow is struck to the patella; and the extent of the projection of the leg is seen to become less and less in proportion as the blows fall higher up.

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[1] "Deutsches Archiv für klinische Medicin," vol. xxiv., p. 178. Leipzig, 1879.

The response to percussion of tendons, fasciæ, periosteum, and muscles has appeared to me to differ in certain particulars according to the nature of the lesion by which it is produced; and when this part of our subject will have been thoroughly worked out, I am convinced that it will be found to possess considerable diagnostic importance.

I propose to distinguish amongst the whole class of exaggerated reflexes :—

1st. The cerebral type;
2nd. The spinal type; and
3rd. The muscular type.

1st. *The cerebral type* is seen where sclerosis of the cord is consequent upon brain lesions, such as tumour, softening, hæmorrhage, and similar conditions. The response is moderately quick and very extensive, so that, taking the patellar reflex as an example, the leg is thrown forward a considerable way up, with a wide swinging motion, and gradually settles down again after some considerable analogous oscillations.

2nd. *The spinal type* is seen in spastic spinal paralysis, insular sclerosis, and combined system diseases of the cord, and is distinguished by an exceedingly quick and jerky motion of the leg, which, however, does not nearly cover the same ground as the reflex of cerebral type. The leg is jerked forward most instantaneously; indeed if one might employ a Hibernicism, I would say, almost before the blow is struck; and the after-oscillations are of the same short and jerky character.

3rd. *The muscular type* is seen in local morbid conditions, such as peripheral paralysis, more especially of the portio dura. The muscular contraction which is then observed is very analogous to that which is seen if a constant current is made to act on a muscle separated as far as possible from all nervous elements. We perceive, then, not a quick jerk of the muscular fibres, but a sort of sluggish wave in the same, which begins slowly, lasts some time, and subsides slowly. There may be a contraction of the entire muscle, but the contraction is generally stronger in those fibres which are directly percussed.

The exaggerated tendon reflexes may be still further increased by the administration of strychnia, and anything else that causes an exaltation of reflex excitability; while they are diminished by bromide of potassium, and anything which has a tendency to subdue that faculty.

It is generally assumed that the anatomical cause of the exaggeration of the deep reflexes is to be found in sclerosis of the lateral columns, and more especially in that portion of these columns which is known as the crossed pyramidal strands (p. 11). This, however, is by no means proved. We know the function of the crossed pyramidal strands to be to carry the orders of the Rolandic convolutions to the muscles of the limbs, and we should therefore rather expect paralysis to be a symptom of their destruction than exaggerated reflexes. The latter may be seen in cases where there is certainly no disease of the pyramidal strands, as, for instance, in certain forms of epilepsy, in acute febrile complaints, such as typhoid fever, in consumption and other wasting diseases. We cannot explain this by assuming the existence of anæmia of the cord, as we find that in the severer forms of anæmia the deep reflexes are not exaggerated. The theory that the reflex excitability of the cord is unduly increased in consequence of the influence of the cortical centres being removed, is also unsatisfactory; for in hemiplegia after cerebral hæmorrhage, where this influence is certainly removed, we find, at least for a month after the attack, that the deep reflexes are not exaggerated, while the superficial ones are either greatly diminished, or altogether gone. It is more probable that there are peculiar connexions of different spinal reflectory centres with various cerebral reflectory centres, and that the changes in the deep reflexes which we observe are owing to changes in the condition and the relations of some of these several centres to each other ; but the exact way in which the exaggeration of the deep reflexes is brought about has not yet been ascertained.

We occasionally meet, in practice, with cases which have not yet been classified, where there is no paralysis, but such an extraordinary exaggeration of the deep reflexes, that paralysis is simulated. The muscular power does not suffer, and sensibility is normal ; yet the increase of the tendon reflexes is such as to impede all active movements. The patient shows all the characteristic features of the spastic gait, with this difference—that he may continue to walk for hours consecutively, without being actually exhausted. He has to make similar efforts to those of a man who is walking in deep sand, and is therefore more easily fatigued than a healthy person. In a bath, however, he may move about without any trouble at all. The rigidity of the extensors then ceases, and his legs may be flexed with ease.

Sensibility does not as a rule suffer in spastic spinal paralysis, except when the malady is very far advanced. It is true that in the beginning of the complaint the patients occasionally complain of shooting erratic pains in the back and legs, but these rarely reach any degree of severity, and are mostly evanescent. There is no anæsthesia or analgesia either in the skin or in the deeper parts. The superficial reflexes are generally normal. There is no muscular atrophy, the faradic and galvanic tests are fairly good, and there is no trouble on the part of the pelvic organs. The urine is generally normal, but occasionally contains an excess of urea and sugar.

Case 77.—In April, 1883, Mr. Bickersteth, of Liverpool, requested me to see an unmarried lady, aged twenty-one, who had for several years past noticed a gradually increasing weakness in her legs, which had rendered her quite helpless. During the last twelve months, however, the disease did not appear to have grown much worse. She had the greatest difficulty in lifting her feet off the ground, to which they seemed to cling, and which they scraped. Indeed, the gait had the true spastic character, and the patient could only make a few steps when supported by others, or when supporting herself by taking hold of the wainscoting or pieces of furniture. There was no muscular atrophy, but rigidity, and the faradic and voltaic excitability of the nerves and muscles was normal. The left leg appeared to be worse than the right. When examined with the dynamometer for the lower extremities, there appeared to be such loss of power in both legs that the patient was unable to move the index at all. The knee-jerk was greatly exaggerated in both legs, showing spinal type (p. 337); and ankle-clonus could be readily elicited. Direct percussion of any portion of the quadriceps caused the leg to be thrown forward. Passive movements caused an increase in the stiffness of the muscles. In the upper extremities the disease was not nearly so far advanced as in the lower. There was a good deal of muscular force, for the patient squeezed the ordinary dynamometer with the left hand to 130°, and with the right to 140°. Both hands, however, were shaky, awkward, and stiff, and almost useless for those finer movements which are constantly required in daily life. All the deep reflexes of the upper extremities were increased, more particularly that of the biceps, and the flexors and extensors of the forearms, as well as of the interosseous muscles. There were no symptoms whatever of hysteria, and there was no history of injury or severe exposure to cold. Sensibility was normal everywhere. Menstruation and the action of the bowels were regular; the patient slept well; but the urine had a density varying from 1032 to 1035, and contained an excess of urea as well as of sugar, the latter amounting, in one specimen, to nearly forty grains to the pint.

Spastic paralysis is mostly a very chronic affection, which may last for many years without apparently much tendency to shorten life. I have never known a patient to die of it. Charcot states that those suffering from this malady, die of phthisis and other affections which have no immediate connection with the spinal disease; while Erb has seen fatal results from extension of the disease to the medulla oblongata, and from blood-poisoning owing to cystitis and the formation of bedsores. Occasionally, however, the course of the disease appears to be more rapid. Thus Hopkins[1] describes the case of a porter, aged twenty-one, who found, shortly after exposure to wet and cold, his legs becoming very weak and tottering. After some months he lost the feeling in the soles of the feet, and the legs were so rigidly flexed that they could not be passively extended. The urine became alkaline and purulent, bedsores formed over the sacrum and the trochanters, the upper extremities became rigid, the temperature began to rise, and the patient died two years after the commencement of the malady. The post-mortem appearances have already been described (p. 52), and the case was certainly a very unusual one.

In a case described by Cahen, the patient, when apparently quite well, suddenly, while walking in the street, lost the power over his legs, and could only half an hour afterwards crawl along to a wall, where he remained standing for another half-hour; after that he could walk again quite well. Two years afterwards he had pain in the leg for a few days, and involuntary urination. This latter disappeared in a week; but increasing weakness now appeared in the legs, with tremor and rigidity. Retention of urine supervened, and the legs became gradually so stiff that he could not sit up in bed; they were strongly adducted. He could eventually only move the toes a little, having lost all power over the hips, knees and ankles. The patient eventually died of a bedsore which had formed.

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[1] "Brain." October 1883.

In the further course of the complaint sensibility is apt to suffer a good deal. Schultz mentions the case of a patient who had lost the sense of temperature, and burnt himself with a hot bottle without knowing it. Sluggishness of, and eventually total loss of power over, the bladder and rectum are also apt to come on after a time, while the sexual desire and power may remain unaltered until a very late period of the illness.

The brain and cranial nerves are generally healthy. I have, however, known temporary double vision to occur in the beginning.

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The *diagnosis* of spastic paralysis is often very easy, and sometimes extremely difficult, more especially with regard to the question whether there is a decided anatomical lesion or not. This difficulty is increased by the circumstance that there is not a single symptom which is actually peculiar to the disease; for the signs of motor irritation which I have described, and which are the most prominent feature of it, may occur wherever there is functional irritation or structural disease of the pyramidal strands, from whatever cause.

It is hardly possible to confound the disease with *tabes*, in which numerous symptoms in the sphere of sensibility are to be found, where the muscles are flabby, and where the deep reflexes show exactly the opposite features to what we see in spastic paralysis.

In *transverse myelitis* from compression or hæmorrhage, the symptoms supervene more rapidly, and show more the purely paralytic character. Rigidity, if it occur at all, comes on at a later period of the disease. Sensibility suffers *pari passû* with motility, and the bladder, rectum and sexual organs are affected at an early stage.

Cases of spastic paralysis which assume the hemiplegic form, will scarcely be confounded with *cerebral hemiplegia.* In the latter the symptoms come on suddenly; there is frequently loss of consciousness and speech; and generally deviation of the tongue and paresis of the lower branches of the portio dura. Moreover, in the later stage of cerebral hemiplegia the leg is almost always more useful than the arm, while in spastic paralysis the leg is always worse than the corresponding arm.

The contractures and palsies which occur occasionally in *hysteria*, do not in general resemble those which we see in spastic paralysis. Hysterical contractures mostly come on suddenly, over-night, after a violent emotion or a convulsive fit, and affect only one group of muscles, while all others are left intact. Thus the flexor muscles of the forearm may become suddenly contracted, so that the hand is violently clenched and pronated; but there are no corresponding symptoms in the other arm, or in the legs. Moreover there are almost invariably other signs of hysteria, such as a highly emotional temperament, a worrying disposition, globus, aphonia, hemi-anæsthesia, dysuria, dysmenorrhœa, pain in the epigastrium, which is increased by pressure, etc.

The aid which we may derive from the dynamometer for distinguishing between sclerosis and *pseudo-sclerosis* has already been insisted upon (p. 329). We must, however, remember that this has not yet been shown to apply to all cases of pseudo-sclerosis, as in some of them the symptoms appear to be almost, if not quite identical with those of actual sclerosis. These cases are even nowadays not unfrequently called hysterical, although there may not be a single symptom of hysteria, and the temperament of the patient is often exactly the opposite of the hysterical. Patients of this kind are often very calm, intellectually gifted, unemotional, most anxious to get well, and have never shown such symptoms as aphonia, globus, dysmenorrhœa, etc. Even the further course of the disease does not invariably lead us to an accurate diagnosis, for although in some recovery takes place, others go from bad to worse, and are no better off for suffering from a mere functional derangement.

Amyotrophic lateral sclerosis is distinguished from spastic paralysis by the occurrence of muscular atrophy in the very commencement of the complaint. Moreover the former affection is more rapid in its course, and generally affects the upper before the lower extremities; and the lesion has the tendency, at a comparatively early stage, to creep up to the medulla oblongata.

It is impossible to distinguish *insular sclerosis* from spastic paralysis when the former affects only the lateral columns of the cord. In general, however, certain symptoms on the part of the brain and the cranial nerves will be discovered, which will lead us on the right track. These are chiefly nystagmus, a slight degree of optic atrophy, drawling speech, vertigo, and failure of intellectual power.

The *prognosis* of spastic paralysis must depend upon the anatomical changes which may be present, and which it is not always possible to ascertain. There may be chronic myelitis, multiple sclerosis, sclerosis confined at one period to the pyramidal strands, with the tendency to extend later in life to the anterior cornua, the posterior columns, etc.; while in other cases there is nothing but functional irritation, which may after a time pass off. Some cases have completely recovered (Erb, Van der Velden, Henck, Schultz, etc.), but this is exceptional where gross anatomical lesions have occurred. In general the disease is apt sooner or later to extend to other portions of the nervous system, and gradually to undermine the vital powers of the patient.

The *treatment* of spastic paralysis has to vary according to the cause of the complaint. Where there are decided syphilitic antecedents, a specific treatment should be resorted to, on the same lines as for tabes (p. 302). On the other hand, where the sclerosis appears to be functional, more especially if the dynamometer does not show any actual loss of power, the Weir-Mitchell treatment by massage and faradisation may prove beneficial. The constant current, administered in the same way as for tabes (p. 310), will generally, however, be found the best remedy. Of medicines, I have found a combination of arsenic, in doses of from two to ten minims of the liquor arsenicalis, and bromide of potassium, in doses of from fifteen to thirty grains, thrice daily, the most effectual. Where the system is greatly reduced, phosphorus, cod-liver oil, malt-extract, and the generous wines of Burgundy and Hungary should be administered. Warm baths, of a temperature of 92° to 100°, should also be given, either daily or every other day. The temperature, as well as the duration of the bath, should be gradually increased, in accordance with individual susceptibility. Warm salt or sea baths answer sometimes better than those of ordinary water.

Nerve-stretching has been practised in this country and abroad with but indifferent results. Southam,[1] of Manchester, has treated a case which was, however, somewhat unusual, by stretching of the left sciatic nerve. There had been, apart from the spasmodic contraction of the muscles and the exaggeration of the tendon reflexes, severe pain in the abdomen and lower extremities, which did not yield to morphia. The tendon reflexes and the muscular rigidity were diminished after the operation, and the pain ceased on the second day, and had not returned six weeks afterwards. The tendon reflexes, however, were as lively as ever after a fortnight. In a case reported by Westphal, the results were disastrous. The extremities became completely paralysed; there was loss of control over the bladder and rectum, and extensive bedsores formed, which it took years to heal. The operation therefore appears to be as little suitable for spastic paralysis as for tabes.

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[1] "The Lancet," October 8, 1881.

CHAPTER XII.

AMYOTROPHIC LATERAL SCLEROSIS.

THIS form of sclerosis was first described by Charcot,[1] and although it resembles in many respects certain cases of ordinary spastic paralysis, the evolution of the disease shows, nevertheless, such peculiarities as to warrant us to look upon it as one *sui generis.*

We have seen (p. 61) that amyotrophic lateral sclerosis has for its anatomical base sclerosis of the crossed pyramidal strands and of the ganglionic cells of the anterior grey cornua of the spinal cord. It is therefore to be looked upon as a *combined system-disease* of that organ. Its causes (p. 129) are, as yet, very obscure.

The first symptom is loss of power in the upper extremities, more particularly in the hands and fingers, and fibrillary twitches in the affected muscles, such as we see them in the more common form of progressive muscular atrophy. "Pins and needles" and numbness are occasionally felt in the arms. Rigidity and contraction supervene after a time in the affected muscles, and deformities of position are noticed. The arm is firmly drawn towards the body, the forearm is semi-flexed and pronated, and the hand and fingers are strongly clenched. Passive supination and extension are impossible without employing an undue degree of force and causing pain. The tendon reflexes are increased, but there is no anæsthesia of any kind. The electric reactions are not invariably the same, as in some cases the so-called "reaction of degeneration" (Erb) or wasting-test is noticed, while in others the remaining portion of the muscles responds normally to faradisation as well as to galvanisation.

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[1] "Leçons sur les Maladies du Système Nerveux." 2nd Série. Paris, 1874.

The second stage of the disease is ushered in, about six or nine months after the beginning of the first, by an analogous affection of the lower extremities. There may also be "pins and needles" and numbness in the legs, but the principal symptoms are again loss of power, twitches of the muscles, with rigidity and exaggerated tendon reflexes. The legs are rigidly extended, and there is tremor on attempting movements. After a time the muscles become atrophied, and there is a proportionate decrease of rigidity and reflex excitability. The bladder and rectum continue to act normally, and there is no tendency to bed-sores.

The third stage supervenes more or less rapidly with symptoms of labio-glosso-laryngeal paralysis; the patient becomes unable to masticate, to swallow, and to speak; respiration and circulation eventually suffer, and death takes place within from one to three years from the commencement of the disease.

Ferrier[1] has reported cases in which the medulla oblongata appeared to be the first to suffer, and where the affection subsequently crept down to the upper and lower portions of the cord. Where the roots of the spinal accessory nerve in the cervical portion of the cord suffer, there may be such rigidity in the trapezius and sterno-mastoid muscles that the head appears completely fixed. In such a case, in which I was consulted in April, 1884, the diagnosis of "ossification of the muscles" had been made! If the temporal muscles are rigid, from the sclerosis affecting the muscles supplied by the minor portion of the fifth nerve, the mouth can hardly be opened. Occasionally there is, in place of common atrophy, pseudo-hypertrophy of the affected muscles, which may render the recognition of the disease difficult.

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[1] "The Lancet," vol. i., p. 822. 1881.

The diagnosis of amyotrophic lateral sclerosis is, however, in general easy. It is distinguished from *spastic paralysis* by its much more rapid course, by its affecting generally the upper extremities previous to the lower, and by being accompanied with muscular wasting at a comparatively early stage. With *progressive muscular atrophy* it can hardly be confounded, as in that malady loss of power and muscular wasting proceed *pari passû*, and there is no rigidity or exaggerated tendon reflexes. Where bulbar symptoms are the first to appear, *labio-glosso-pharyngeal paralysis* may be suspected, but the speedy supervention of signs of motor irritation in the upper and lower extremities will generally be sufficient to lead us to an accurate diagnosis.

The prognosis of amyotrophic lateral sclerosis is unfavourable, as until now we have not found any remedy which seems in any way capable of arresting the rapid downward course of the disease. The constant current and prolonged warm baths promise more than any other remedies.

CHAPTER XIII.

SECONDARY LATERAL SCLEROSIS.

THIS form of sclerosis, the anatomical features of which have already been described (p. 56), is owing to destructive lesions of the motor sphere in the brain or spinal cord, and as it always occurs below the seat of the anatomical lesion, is also often called *descending lateral sclerosis.* The part affected is not the entire lateral column, but only that portion of it which is known as the crossed pyramidal column. In cerebral lesions, the opposite side, and in lesions of the cord, if they are unilateral, the same side, is thus affected.

By far the most frequent causes of secondary lateral sclerosis are hæmorrhage in the central ganglia of the brain, and embolism or thrombosis of the middle cerebral artery and its branches. If hæmorrhage or softening in the central ganglia be very limited in extent, it may partly destroy or displace the grey nuclei, but does not tear up the white internal capsule, which constitutes the great conducting path of motor power from the brain to the limbs. In some cases, indeed, the pathological lesion is so slight that there are hardly any symptoms during life; but where the lesion is somewhat extensive, crossed incomplete hemiplegia is the result, which is generally transitory, for the patient mostly recovers the power over the affected side to a great extent in a few weeks or months. In such cases no secondary or descending sclerosis of the pyramidal strands takes place. But where hemiplegia remains more or less permanent, and is after a time followed by late rigidity of the paralysed muscles, the lesion is known to have invaded that portion of the internal capsule which contains the pyramidal strands, thus severing the connection between the psychomotor centres or Rolandic convolutions and the extremities.[1]

It seems at first sight singular that disease of the grey nuclei should cause less severe symptoms than destruction of white matter, which does not generate, but only conduct power. This fact, however, is easily understood if we consider that there are several grey centres, and only one white conducting strand. The principle of compensation or substitution applies to the several grey nuclei. Where the influence of the lenticular nucleus is removed, the patient may fall back on the caudate nucleus and the hemispheres, and will after a time be found not to be much worse off than he was before the attack. Destruction of the internal capsule, however, creates a chasm between all the grey centres generating motor influence, on the one hand, and the portio dura and the extremities on the other hand, which cannot be bridged over, and invariably leads to sclerosis of the white conducting strands behind the lesion, which generally becomes developed a month or two after the stroke.

The following symptoms accompany this descending degeneration :—About a month or six weeks after the stroke, there is a feeling of stiffness, which differs from the powerless feeling of the first few weeks, in the flexor muscles of the paralysed forearm, and these gradually become rigid. The fingers are clenched, and the contraction is sometimes so extreme that the nails cut into the flesh. The thumb is flexed, and so strongly adducted that it disappears under the other fingers. These latter offer great resistance to an attempt to open the hand of the patient. The forearm is pronated, the elbow semi-flexed, and the arm adducted to the body. A degree of contraction, however, is present in all the muscles—the extensors and abductors, as well as the flexors and adductors, and this accounts for the circumstance that it is sometimes difficult to make any considerable change in the position of the limb by passive movements.

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[1] *Vide* my Lecture, "On the Pathology and Treatment of Cerebral Paralysis." "British Medical Journal," June 4th and 11th, 1882.

In the lower extremities the muscular rigidity is generally less marked. We notice it, however, in the hamstring muscles of the thigh and the flexors of the leg; and where this reaches an extreme degree, it renders walking quite impossible, as the leg is then flexed on the thigh, and the thigh on the pelvis, so that the heels touch the buttocks. If the extensors and adductors of the leg are thus affected, the appearance of pes equinus is caused. The patient then walks on tiptoe, and, in order to diminish the dragging of the foot on the ground, instinctively swings the leg from without inwards, which constitutes a peculiarly characteristic kind of gait.

There is also a certain amount of rigidity in the face, which is more particularly noticed when the patient is talking, laughing, or crying. While at first, when there is simple palsy, the commissure of the lips is depressed, it becomes later on raised on the paralysed side. The naso-labial sulcus appears more marked; the nostril is dilated; and the eye looks smaller, from rigidity of the orbicularis.

Muscular stiffness is greater when the limbs are cold than when they are warm. For this reason, the hand appears more open when the patient is in bed, and the arm is kept under the bedclothes, than when he is up. It is also greater in the waking state than during sleep; and this is no doubt due to the circumstance that emotions have a tendency to increase it, so that their absence during sleep renders the contraction less marked. The rigidity is also exaggerated by efforts to use the paralysed muscles; by injuries, such as a blow or a fall; by faradisation of the skin ; by the old-fashioned method of using electro-magnetism, which consisted of making the patient take hold of two metallic handles with both hands, and thus sending a powerful current right through the body. Duchenne has related the case of a student who treated himself in this fashion, and in whom the exciting effect was so great as to cause a fresh paralytic attack. The administration of strychnia, which was formerly much in vogue for the treatment of this form of paralysis, likewise tends to increase the rigidity ; and, if it be pushed so far as to cause convulsive movements in the muscles, they are more violent in the paralysed than in the healthy side. Anything and everything, therefore, which has in a general way the tendency to exaggerate the reflex excitability of the cord will increase the rigidity of the paralysed muscles.

Late rigidity of the paralysed muscles after hemiplegia was formerly believed to be owing to inflammation of the brain at the seat of the lesion, which was thought to follow habitually upon an attack of apoplexy ; but the changes which really follow an effusion of blood are not of an inflammatory character. Nor is it due to the shrinking of the cerebral cicatrix, as Todd believed ; nor to peripheral neuritis, which has been assumed to occur by other observers. Neither can we explain it by an increase of reflex excitability, owing to the removal of the inhibitory influence of the brain ; for the influence of the brain upon the paralysed limbs is removed immediately after the attack, while rigidity only comes on a month or two afterwards.

Pathological anatomy shows as the only constant alteration in these cases descending sclerosis of the crossed pyramidal strands ; and we, therefore, assume that this sclerosis leads to irritation in the large motor cells of the anterior horns of the spinal cord, and thus causes that contracted condition of the muscles which is in reality an exaggeration of their normal tonicity, this latter being under the direct influence of those ganglionic masses. Irritation of the horns, however, is a very different thing from actual disease or destruction of them; and it is only in exceptional cases of cerebral paralysis that these horns become really diseased in the further course of the affection.

In the vast majority of cases there is no muscular wasting, except what may be accounted for by disuse. It is indeed singular to see patients who have been deprived of the power over one side of the body more or less completely for five or ten years, and more, and in whom nevertheless the muscular tissue is fairly nourished, and where faradisation causes at once a ready response by inducing electro-muscular contractions. Compare with such cases those of infantile paralysis, where the muscles waste away, and lose their faradic excitability within a few weeks, and an enormous difference is at once seen, which is, however, satisfactorily explained by the anatomical data which I have just given.

In some cases, however, the sclerosis spreads from the lateral column to the anterior horns; and we then notice a corresponding change in the symptoms. As the centre of tonicity is gradually being destroyed, the rigidity of the muscles diminishes *pari passû*, and ultimately vanishes altogether. When the muscles become more relaxed, the patient is apt to think that he is getting better; but such hopes are delusive, for the limbs become, on the contrary, more feeble as time goes on, and the muscles are found to lose their faradic excitability. This goes on much in the same order as we see it in progressive muscular atrophy, affecting first the muscles of the shoulder and the hand, and afterwards other groups of muscles in succession. It is generally protracted over two or three years; and shooting pains are often felt in the muscles when the wasting commences. All grey cells being connected, the degeneration may spread from the anterior to the posterior horns, or to the anterior horns of the opposite side by means of the anterior commissure; and we have then no longer hemiplegia, but paraplegia with muscular atrophy.

It is doubtful whether cases of permanent hemiplegia ever exist without at least some degree of rigidity. This does not of course apply to lesions of the grey nuclei, where the internal capsule remains unaffected; for in these cases the paralysis mends so much soon after it has been established, that we can hardly call them hemiplegic two or three months after the attack, when there remains simply a degree of awkwardness in using the fingers, but no actual paralysis. The patient then uses his Rolandic convolutions where formerly he used the lenticular and caudate nuclei; and more conscious attention and trouble is therefore required than before the stroke. Where, however, the internal capsule has been injured, and the affection is, therefore, more severe, there is probably always at least some degree of contraction, which is sometimes, indeed, barely appreciable when the limb is quiet, but which becomes at once obvious when the patient is requested to make an effort, however slight. Thus, for instance, he may be able to move his arm and hand in all directions; but when told to pick up a pin, or to unbutton his coat, the fingers and hand are seen to contract, rendering such manipulations difficult or impossible. On examining the state of reflex excitability, that of the tendons is found to be exaggerated. The time of menstruation in women is particularly favourable for the production of these phenomena—no doubt owing to the excitability of the cord being abnormally high during that period.

The prognosis of this form of sclerosis depends upon the amount of damage done, either by softening or hæmorrhage in the brain. Lesions of slight extent may be more easily repaired than those occupying a large area. Age and constitution of the patient have that influence which must be accorded to them in all diseases; and, finally, much depends upon the treatment which is adopted at an early stage of the malady, and chiefly before sclerosis has been fully established. With regard to this point, the occurrence of ankle-clonus after an apoplectic or paralytic seizure should be carefully watched for, as it is a diagnostic sign of the utmost value, leading us to predict the probably speedy appearance of late rigidity in the paralysed muscles; and, as soon as that symptom has made its appearance, no time should be lost in resorting to special therapeutical measures.

The *treatment* of cerebral paralysis would, *primâ facie*, appear to have but slight chances, as in one class of cases there is a natural tendency to recovery of function by compensation of allied structures, while in another the destructiveness of the lesion and its consequences would seem to render all therapeutical efforts futile. The question of treatment has, therefore, been disregarded by many of those who have been most forward to investigate the pathology of this condition. Nevertheless this must, for all time to come, be the chief point of interest for the practitioner. No doubt some cases are incurable; yet I think I shall be borne out by those most conversant with this subject when I say that few cases are met with in practice which may not be improved by judicious treatment.

The remedies I have used for this special purpose have been: first, phosphorus, sometimes simply dissolved in a sufficiency of oil, and administered in a capsule or perle twice a day, in doses of one-thirtieth of a grain, and sometimes in cod-liver oil, where the general state of the patient seemed to render the latter medicine advisable; and second, the constant current, sent transversely through the brain by one electrode being placed to the right and the other to the left mastoid process, and also longitudinally by one being placed to the forehead and the other to the occiput. The cathode, which is the more stimulating of the two poles, should be nearest to the seat of the lesion; so that, for instance, in right hemiplegia, the cathode is placed to the left, and the anode to the right mastoid process. Such applications are made daily, the current being allowed to pass for from five to ten minutes at a time. Later on, local applications of the current to the paralysed parts are combined with this. These latter are not so much intended to act on the motor nerves and muscles as on the sentient nerves, by which the stimulation is reflected to the suffering centre. Faradisation is also sometimes useful, and should be employed to the antagonists of the contracted muscles.

Our second object should be to counteract the sclerosis which is apt to follow the attack. For this we have several remedies, which, if used at a sufficiently early stage, have appeared to me useful. These are principally the salts of gold and silver and ergot of rye. Gold is given either as chloride of gold or, better still, as chloride of gold and potassium, in doses of one-eighth to one-half a grain, in pills. Silver is given as the oxide, nitrate or phosphide, in doses similar to those of gold. Ergot of rye I generally give in the form of the liquid extract, in doses of from half a drachm to a drachm three times a day. A good plan is to give a gold-salt for a month, a silver-salt for a month, and ergot for a month. That remedy under which most improvement appears to take place may afterwards be continued, according to the special features of the case.

Iodide of potassium does no good in embolic softening, but appears useful in promoting the absorption of the clot after hæmorrhage. This pathological process is generally finished in about six weeks after the attack, and it is rare to see much good resulting from that remedy at a later stage of the disease. Bromide of potassium, on the other hand, is useful when reflex excitability appears much increased.

A word must be said about the employment of strychnia in these cases. This is a remedy for paralysis hallowed by tradition ; yet nothing could well be more unphilosophical, or indeed hurtful to our patients than to sanction or to continue such a practice. Strychnia has the tendency to increase the exalted state of excitability in the spinal cord, and thus to render the muscular rigidity worse. It is, therefore, a medicine which should never, on any account, be given in such cases.

The general health has, of course, to be carefully attended to at the same time. We must remember that some patients, viz., the subjects of embolism, suffer from heart-disease ; while others, in whom the attack has been owing to hæmorrhage, are probably affected with small aneurisms in different portions of the brain, which may burst subsequently, and give rise to fresh attacks. Straining at stool is dangerous for such persons, and the action of the bowels should therefore receive great attention. The *morale* of the patient is often improved by change of air and scene, as soon as he is sufficiently well to bear a journey. We cannot expect much from mineral water cures in the further progress of these cases. Wildbad, Gastein, Buxton, and other spas have had a reputation in the treatment of cerebral paralysis and secondary sclerosis which has, however, not stood the test of time and strict criticism ; and it is, on the whole, better to send such patients to places which are easily accessible, where all comforts can be procured, and which possess well-known climatic advantages. A moderately dry bracing air is found to be particularly suitable for these patients ; but high elevations must be avoided.

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There is a peculiar form of secondary sclerosis, causing *spastic paralysis in children*, to which I have already alluded (p. 57), and which is owing to disease of, or deficiency in, the Rolandic convolutions.

The symptoms of this form of sclerosis point to the circumstance that the paralysis is primarily dependent on a cerebral lesion, and that the spinal cord, or rather the pyramidal strands of the lateral columns, are only subsequently affected.

In some cases there is original malformation of the skull, which may resemble that of a microcephalic idiot; and there is a distinct deficiency of cerebral matter, either in one or both hemispheres (porencephaly). In other cases, protracted or instrumental delivery is the cause. The head is sometimes so much squeezed by the blades of the forceps that a crushing lesion of the cerebral substance is produced, which is followed by encephalitis; or there is hæmorrhage in the cortex. The infant is then seized with convulsions immediately after birth, and may continue convulsed for some days, and is soon after discovered to be hemiplegic; or it is seized with convulsions a few months or even a few years after birth. The convulsive seizures may be of the ordinary epileptic type, viz., unconsciousness and general convulsions, or only one-half of the body may appear convulsed. Sometimes there are five or six such fits, and the child is afterwards found to be hemiplegic. The hemiplegia becomes after a time complicated with late rigidity, and occasionally with hemichorea and athetosis. The development of the paralysed limbs is more or less arrested; the bones are seen to be shorter and smaller than those of the healthy side. There is, however, no muscular wasting, such as we find after an attack of polio-myelitis; and the faradic and voltaic responses are normal. In some cases there is no actual paralysis, but paresis and great awkwardness in the use of the limbs. The tendon reflexes are invariably exaggerated; and the rigidity of the muscles is sometimes so great that the legs are in a position of extreme adduction and extension The paralysis is either hemiplegic or paraplegic, and if the latter, it must be looked upon as bi-hemiplegic and cerebral. Occasionally wasting of the rigid muscles supervenes, and we have then the clinical aspect of a combined secondary system disease of the cord.

There is always a degree of mental deficiency in such children varying from dulness and stupidity to complete imbecility. The speech is drawling, or peculiar in other ways. Children of this kind sometimes use words and have a pronunciation which is quite their own, and retain them, if they survive, for the rest of their lives. Some do not walk at all; others begin to walk when they are six or seven years old, and retain throughout life a peculiarity in their gait. We find occasionally nystagmus, convergent strabismus, inequality of the pupils and protrusion of the eyeballs. Convulsive fits may come on again at any subsequent period.

The prognosis of spastic paralysis of children is most gloomy. Orthopædic surgeons generally treat these patients with more zeal than discretion, by cutting tendons and the use of complicated apparatus; but in the majority of cases such interference can only injure.

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Secondary descending sclerosis after certain affections of the spinal cord is so overshadowed by the symptoms of the primary disease that it is not necessary to dwell upon it in detail. It occurs in hæmorrhage from injuries and after crushing lesions of the spinal cord, provided the patients survive long enough for secondary degeneration to become established. I saw a case of this latter kind (No. 78), in consultation with Dr. Giffard, of Egham, on December 5th, 1883. It occurred in one of the pupils of the Royal Engineering College at Cooper's Hill, in consequence of an accident while playing at football, on November 29th, when the lad, aged nineteen, was thrown heavily on his back, with the result of becoming immediately completely paralysed and anæsthetic from the waist downwards, but not insensible. There was complete paralysis of the bladder and bowels, and a moderate degree of priapism. He was unable to change the position of his body, and there was also paralysis and anæsthesia of the forearms and hands, while a feeble degree of motion and sensation existed in the arms and shoulders. He was unable to cough, to clear his throat, to sneeze, or blow his nose; but could move his head from one side to the other, and had no difficulty in speaking, masticating, and swallowing, and could put out his tongue. I found on examination that all the superficial and deep reflexes of the limbs and body were completely lost. This rendered it evident that there was a crushing lesion of the cervical portion of the spinal cord, and that the grey centre of it had been most gravely injured. I therefore predicted the occurrence of rapid muscular atrophy in the paralysed limbs, supposing the patient were to survive the injury. When I saw him again on January 30th, 1884, extreme atrophy of the muscles of the body and all four extremities had become established, while *the few remaining muscular fibres were in a state of rigidity, and showed increased tendon-reflexes,* showing that secondary degeneration of the pyramidal strands of both sides had become developed.

Similar appearances are noticed in acute transverse myelitis which is likewise owing to injury, and in which, if the patient survives sufficiently long, secondary sclerosis below the seat of the disease is apt to supervene.

CHAPTER XIV.

SCLEROSIS OF GOLL'S COLUMNS.

SECONDARY sclerosis of Goll's columns is almost invariably one of the later lesions of tabes and of transverse myelitis. Primary sclerosis of the same strands, apart from any other lesion, appears to be exceedingly rare, as up to the present time only three such cases have been recorded, viz., by Pierret,[1] Ducastel,[2] and Gowers.[3] The symptoms appear to have been a mixture of those of tabes and spastic paralysis ; but this is a chapter of pathology which has yet to be written.

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[1] "Archives de Physiologie," p. 74. Paris, 1873.

[2] "Gazette Médicale de Paris," No. 4. 1874.

[3] "The Lancet," vol. ii., p. 876. 1879.

CHAPTER XV.

MULTIPLE OR INSULAR SCLEROSIS.

We have seen the anatomical features of multiple, insular, or disseminated sclerosis, or sclerosis in patches, to consist of multiplication of the nuclei and proliferation of the fibres of the neuroglia, followed by degenerative atrophy of nerve-tubes, with persistence of the axis-cylinder (p. 62). Its causes are still very obscure, and no age or sex is exempt from it (p. 127). As Charcot was the first to distinguish this form of sclerosis as a separate pathological entity, I have elsewhere[1] proposed to call it "Charcot's disease."

The commencement of multiple sclerosis may be sudden, with an attack of apoplexy or epileptiform seizures. If the former, the symptoms may be almost identical with those of an ordinary attack of apoplexy from cerebral hæmorrhage; that is to say, there is a feeling of giddiness and confusion, with noises in the head and tingling in the limbs, which is, after a variable time, followed by loss of consciousness and coma; the pulse is accelerated; the temperature may rise to 103° or even 105°; the face is livid and swollen; there may be involuntary evacuation of the urine and fæces; and hemiplegia is discovered. After a time, however, which varies from twelve to forty-eight hours, the patient begins to rally; he slowly regains his consciousness, and within a few days likewise the power over his side. Such attacks may come on every few months, with the same result; but the patient may die in one of them. They are most probably owing to ischæmia of the brain from vasomotor spasm ; for in fatal cases neither congestion nor embolism nor hæmorrhage has been found to account for the symptoms, while old lesions in the medulla oblongata, affecting the centre of vaso-motor power, have generally been discovered. Sometimes the patient does not recover completely from such an attack, but is found afterwards to be troubled with double vision, amblyopia, nystagmus, impaired articulation, and other symptoms of defective cerebral nutrition. The patient whose case (No. 51) has been described on p. 144 had had repeated attacks of hemiplegia and aphasia *for five years* consecutively before the symptoms of multiple sclerosis of the cord became definitely established. The occurrence of such attacks always raises a strong presumption of syphilis being at the bottom of the complaint.

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[1] "Diseases of the Nervous System," p. 330. London, 1877.

In the majority of cases, however, the invasion of the disease is slow and insidious ; and we may, as in tabes, distinguish three several stages or periods which may, and generally do, merge imperceptibly into one another.

In the first period the symptoms are often ill defined. The principal complaint of the patient is that of a gradual *loss of power* in one or both lower extremities, which has the tendency to become aggravated as time goes on, and to spread to the upper extremities. The limbs feel heavy, are difficult to move, and spastic gait (p. 332) is common. Muscular stiffness, rigidity, and contractions appear occasionally at a somewhat early period of the malady, which is therefore apt to be confounded with spastic paralysis; but at this period the patient is still able to go about and attend to his avocations. He frequently, however, complains of headache, giddiness, and mental depression; the memory is impaired; there is great indifference to the affairs of daily life, and occasionally melancholia, with refusal of food. Grandiose delirium and other symptoms of general paralysis of the insane have been observed.

Where the patients are children, we notice an inequality in their temper, and a degree of excitability which is unusual. There is often immoderate laughing or crying about nothing at all. The mind appears dull and clouded; the memory, which may formerly have been excellent, becomes impaired; and the child eventually appears imbecile. In such cases we have no doubt to do with sclerotic patches in the cineritious substance of the hemispheres. Convulsions occasionally usher in the beginning of multiple sclerosis, as of so many other infantile complaints; and Bristowe has seen a case in which somnambulism occurred.

Symptoms in the sphere of *sensibility* are on the whole rare. Lightning-pains are generally absent, but occur now and then (Case 49, p. 143), as well as paræsthesia of different kinds; and there may even be circular tightness or belt-sensation, plantar anæsthesia, and numbness in the sphere of the ulnar nerve. In such cases ataxy of gait, or a gait which appears to be a mixture of ataxy and paresis (Case 48, p. 142), may be seen. There is decided loss of power, combined with jerked movements of the legs and stamping of the ground, as well as Romberg's symptom; so that the resemblance to tabes is close. In such cases we assume the existence of sclerotic patches at various levels of the posterior columns, in addition to those which we have reason to suspect in the antero-lateral columns; yet such patches have occasionally been found in the posterior columns post mortem, where there had been no symptoms in the sphere of sensibility during life. Schüle[1] explains this by assuming that certain portions of the posterior columns and the central grey matter may still be able to conduct sensitive impressions, even after considerable damage to other portions of the same tracts has been done. In cases of combined system-diseases of this sort, the symptoms incline towards that column which is principally affected. Where, for instance, patches exist throughout the extent of the posterior columns, including the posterior root-zones of the lumbar enlargement, there will be more the symptoms of tabes, with loss of deep reflexes, and flabby muscles; while if the whole extent of the lateral column is more or less affected, and patches exist only here and there in the posterior columns, we shall have more the symptoms of spastic paralysis, with rigid muscles and exaggeration of the deep reflexes.

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[1] "Deutsches Archiv für klinische Medicin," vol. vii., p. 159, and vol. viii., p. 223.

The most peculiar and characteristic symptom at this stage of multiple sclerosis, however, is a peculiar kind of *tremor*, which Charcot was the first to notice and to describe. This tremor only becomes manifest when somewhat extensive purposive or intentional movements are made, and ceases completely during rest. Restricted movements are possible without tremor; or the shaking is so slight that it can only be distinguished in such acts as writing, where, even in the beginning of the disease, no plain and bold strokes are possible, but where there is in almost every letter that is written an evidence of slight unsteadiness, as if the person who was writing were under the influence of drink.

Sclerotic tremor has the peculiarity of being rhythmic in character, there being a succession of muscular contractions occurring at more or less regular intervals, and keeping, on the whole, tolerably close to the direction which it is intended to follow. The keener the intention to carry out a certain movement, and the closer the attention given to the performance, the greater is the tremor. On this account, the patient has generally less difficulty when he is alone and unobserved, than when examined by a doctor. If the patient be requested to put out his tongue, that organ is jerked forward suddenly and withdrawn again, and is seen to work about either in or out of the mouth, together with tremor in the labial and other facial muscles. Similar shakings are seen when the patient is asked to move his arms or legs, or to sit up or stand up, and more especially when complex movements are attempted. The tremor frequently affects other parts than those which happen to be active; so that when the arm is moved, the head and body begin to shake; and if the patient is requested to walk, no part appears quiet, but he is shaking all over.

Sclerotic tremor interferes, therefore, more especially with all the ordinary useful complex movements of daily life. The patient is unable to take a cup of tea without spilling it; he has the greatest difficulty in dressing, shaving, buttoning his clothes, in carving a joint or cutting his meat, in writing, playing the piano, etc. The character of the handwriting, more especially, becomes completely altered, and, after a time, anything that the patient may attempt to write, is quite illegible.

The tremor is generally more pronounced in the limbs than in the body; but I have seen a case, which I believe to be unique, where sclerotic tremor affected the muscles of the body exclusively without any others.

Case 79.—This was the case of a girl, aged 25, who had been pronounced to suffer from hysteria, but in whom not a single symptom of hysteria existed when I examined her. The girl was of an exceptionally calm and unemotional temperament, and deeply regretted being disabled by her affliction from supporting her parents, who were old and infirm. She showed no symptoms whatever which could have been referred to sclerotic patches in the pons, medulla oblongata, or other portions of the brain; spoke in a perfectly natural manner, could protrude her tongue without jerking it about, and had no nystagmus. She could use her hands for work, writing, doing her hair, etc., and had no difficulty in moving her legs in bed. As soon, however, as she attempted to rise from the horizontal position, which she had habitually assumed for the last six months, sclerotic tremor of such a violent character commenced in all the muscles of the body as to render all efforts for getting up unavailable. The body of the girl was then swayed backwards and forwards in the most extraordinary manner, the jerks being short and sudden, and succeeding each other rapidly. Respiration became panting, and the pulse accelerated, but the muscles of the head and the limbs did not participate in the tremor. This same kind of tremor also occurred when her attendant attempted to shift her position ; and it was of such a distressing character, that the patient preferred remaining for many hours consecutively in exactly the same position to being shifted, in spite of the discomfort entailed upon her by her fixed attitude. All the deep reflexes were exaggerated ; but there were no other symptoms whatever, either in the sphere of sensibility or elsewhere. In this case the anatomical lesion must have consisted of sclerotic patches confined to the antero-lateral columns of the entire dorsal and the lower cervical and upper lumbar portion of the cord.

From this description it will be seen that sclerotic tremor and the tremor of *paralysis agitans* are of an entirely different nature. In shaking palsy, or Parkinson's disease, the tremor occurs chiefly during rest. If the hand of the patient be laid on a table near which he is sitting, or on his thigh, rhythmic oscillations are observed, which go on incessantly at a steady rate, generally between eighty and a hundred in the minute, as long as the hand remains in the same position ; but if the patient moves his hand or arm, or takes hold of an object, the tremor ceases for the time being altogether. These peculiarities are in most cases so well marked, that no difficulty can arise in distinguishing the tremor of shaking palsy from that of multiple sclerosis. There are, however, exceptional instances, in which the symptoms are so mixed, or apparently muddled up, that there is a difficulty in deciding to which disease the tremor belongs.

Again, the movements of *chorea* in no way resemble sclerotic tremor. In chorea the movements are disorderly and without purpose, and occur during rest as well as motion. The utter absence of intention is their chief characteristic feature. Thus, when a patient affected with chorea carries a cup of tea to his mouth, we notice movements of an entirely contradictory character, which have the tendency to counteract rather than to assist the intended movement; while in multiple sclerosis the intended direction of the movement persists in spite of the impediments which are occasioned by the tremor.

In the ataxic and terminal stages of *tabes spinalis* we notice movements devoid of co-ordination, which in a measure may resemble choreic and sclerotic movements; but there is in them no real tremor or oscillations, and they are at fault rather by being too abrupt and extensive, and therefore devoid of order. The ataxic movements again are distinguished from choreic and Parkinsonian, by not occurring during rest, but only when movements are attempted. The want of co-ordination in the tabid is moreover always increased when he closes the eyes.

Charcot has explained the sclerotic tremor by assuming that in these patients the nervous influence is only transmitted by the axis-cylinder, which is deprived of its medullary sheath, so that there is no continuous action, but jerky and irregular oscillations. This view, however, appears somewhat too mechanical; and most observers are now inclined to believe that the tremor is rather owing to localisation of the sclerosis in certain parts above the medulla oblongata and the pons Varolii, as it appears to be absent where the patches are confined to the cord, and, on the contrary, present where areas of sclerosis are found in the central ganglia and other portions of the brain without simultaneous disease of the cord. The matter, however, is still very obscure; and Bastian[1] has recently recorded a case in which the tremor was absent from first to last, and where the autopsy showed numerous patches in the pons and medulla oblongata, one of them being a quarter of an inch in diameter, while others varied in size from that of a pea to a mustard-seed. The white substance of both hemispheres also showed small grey areas, none of which, however, implicated the grey substance of the cortex. Patches were also found on the surface and through different parts of the interior of both thalami, while the corpora striata and the cerebellum were free of them. The cord showed, after straining, small areas of degeneration in different portions of the lateral and posterior columns and the contiguous grey matter. Bastian has suggested that the absence of tremor in his case may have been owing to there having been, at an early period, considerable degeneration in the anterior pyramids, cutting off the cerebral influence from below; but this explanation appears far from satisfactory. It is true that there was an early affection of the medulla oblongata, as shown by the presence of drawling speech; but at this time the cerebral influence was by no means cut off, since the patient was still able to walk and stand, and to use his hands. While therefore variations in localisation are probably the cause of either absence or presence of tremor, the exact spot which is of influence in the production of this symptom has not yet been determined.

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[1] "Clinical Society's Proceedings," "British Medical Journal," Oct. 20, 1883.

Strümpell has suggested that the tremor of multiple sclerosis is owing to a pulling and stretching of tendons, which takes place as soon as a sudden or energetic movement is attempted, and that this causes reflex movements in the muscles corresponding to those tendons which interfere with the proper execution of the intended movement. If, for instance, the forearm be quickly flexed, there will be a contraction in the triceps, causing the forearm to be stretched for a short time, and thus the intended movement will appear irregular and tremulous. This explanation, however, appears likewise unsatisfactory; for if we were to accept it, we should expect to find sclerotic tremor even more marked in spastic paralysis, where, however, the tremor or trepidation which is seen is of an entirely different character.

A peculiar form of *vertigo* is frequently observed. All objects seem to the patient to be spinning round, and he himself with them ; so that in order to save himself, he takes hold of anything near him. This giddiness is apt to come on in attacks of variable duration, and must not be confounded with vertigo of an entirely different character which occurs through paralysis of one or several of the ocular muscles and consequent double vision. The latter may be at once arrested by closing the affected eye.

Drawling speech is very common, and may be the first symptom of the illness (Case 42, p. 127) ; articulation is slow and hesitating ; one syllable is pronounced after the other in a sort of rhythmic manner, making the delivery exceedingly monotonous. Occasionally even one syllable is drawn out to an enormous length, so that the patient does not say "yes," but "y-e-e-e-e-es." At the same time there is no modulation in the voice, the pitch of which is somewhat high, and remains exactly the same throughout a conversation. After a time, however, the speech becomes so indistinct as to be unintelligible to a stranger. The difficulty is, however, always of the anarthric rather than of the aphasic type, as the patient seems never to be at a loss for words to express himself, and is able to finish his sentence without difficulties on that score. The impairment of articulation is presently followed by other symptoms showing an affection of the medulla oblongata, viz., dribbling of saliva from one or both corners of the mouth, great trouble in mastication and deglutition, and feeble phonation. The tongue may at this time be still freely movable, but often shows fibrillary twitches.

Where multiple sclerosis occurs in children, their speech is generally incomprehensible from the first.

Affections of the ocular muscles are by no means un common. There may be convergent strabismus, nystagmus, or ptosis ; and some of these symptoms may be the first to attract attention :—

Case 80.—In October, 1880, Mr. Power requested me to see a gentleman, aged twenty-nine, single, who had had syphilis in 1872 ; and in May, 1879, was troubled with double vision, owing to paralysis of the rectus internus of the left eye. He underwent treatment at home by iodide of potassium, iron, and strychnia without any effect, and then went to Aix-la-Chapelle, where he had a large number of inunctions, together with applications of the constant and induced current. In spite of this treatment, however, the disease of the third nerve gradually progressed so as to develop into an incomplete form of ophthalmoplegia. When I saw him, there was ptosis of the left eyelid ; almost complete paralysis of the left rectus internus, and paresis of the rectus superior. The eye could not be brought into the inner corner, but could be slightly moved upwards, although not nearly to the same extent as the other one. The pupil was enlarged, and did not constrict by ordinary daylight, although it contracted sluggishly when a burning match was approached to the eye. There was no affection of the bowels, bladder, and sexual organs. The knee-jerk was found to be greatly exaggerated on both sides. I saw the patient again in May, 1884, when paresis of the bladder, with tendency to ammoniacal decomposition of the urine, impotency, and difficulty in walking had become developed, and there was slight sclerotic tremor in the upper extremities, with exaggeration of deep reflexes. The muscles of the lower extremities were extremely thin and flabby, as is seen in tabes, but without evidence of actual pathological wasting, as after poliomyelitis, or rigidity, as in spastic paralysis.

Nystagmus is sometimes an early, and at other times a late, symptom. In some cases there are two or three oscillations of the eyes in a second, while in others there are only a few oscillations every five or ten seconds. It is, on the whole, more noticeable when the patient fixes his eyes on an object which is moving; and it has appeared to me to partake often of the character of sclerotic tremor.

Amblyopia, with limitation of the visual field, and Daltonism, are important symptoms in multiple sclerosis, more especially when combined with decided ophthalmoscopic changes.

Case 81.—In November, 1880, Dr. Andrew, of Shrewsbury, requested me to see a single lady, aged twenty-six, who had for about eleven years suffered from loss of power which commenced very insidiously in the right leg. After a time the left leg was also affected, so that she had great difficulty in walking; and the disease then gradually crept upwards, invading the upper extremities. Both hands are now very useless, the right more so than the left. The squeezing power, as measured by the dynamometer, is only 30° for the right and 45° for the left hand. There is no sclerotic tremor, but the deep reflexes are considerably exaggerated, while the superficial ones are absent. There is no muscular rigidity or contraction anywhere; the muscles are flabby and thin, but not pathologically wasted, and respond well to galvanisation and faradisation. The knee-jerk is exaggerated, and more so in the right than in the left side. The patient has never had any pain, and there is not a single symptom in the sphere of sensibility. She has had attacks of incontinence of urine. The left optic disc is white, and the arteries are small. There is dimness of sight, and temporal limitation of the field of vision on that side. Both pupils are large. No cause of the affection could be ascertained.

In this case the diagnosis could not be doubtful. It was evidently not a case of tabes, as the patellar reflexes were exaggerated, and sensation was normal everywhere; nor one of spastic spinal paralysis, as the muscles were flabby instead of rigid, and no contracture existed anywhere; nor one of hysteria, as the degree of exaggeration of the deep reflexes varied in different parts; while pain or tender spots of any kind had been absent throughout the illness, and ophthalmoscopic changes existed.

Amblyopia, more especially if connected with ophthalmoscopic changes, may occasionally enable us to distinguish multiple sclerosis from hysteria or pseudo-sclerosis. Gnauk[1] has examined this point in fifty cases of undoubted multiple sclerosis, in most of which the diagnosis was confirmed by inspection; and found, independently of palsies of ocular muscles, inequality of pupils, etc., in twenty-eight cases failure of sight: in eight of these there was simple amblyopia; in five others, diminished activity of vision and limitation of the visual field; and in fifteen, changes in the fundus oculi. The limitation of the field of vision was generally temporal. Amongst the changes in the fundus as seen with the ophthalmoscope, there was total optic atrophy in two cases, partial atrophy in ten, and fresh optic neuritis in three. Optic atrophy may be an initial phenomenon of multiple sclerosis, and may precede the evolution of other symptoms of the disease for years; and there may be no apparent proportion between the ophthalmoscopic appearances and the subjective symptoms complained of by the patient.

While in tabes amblyopia generally merges into amaurosis, with complete atrophy of the optic nerves and total blindness, the amblyopia of multiple sclerosis is not nearly so progressive in character, and often remains stationary for years. This is, according to Charcot, owing to the degeneration being more interstitial than parenchymatous, and to the persistence of the axis-cylinder in the optic nerve-tubes.

Symptoms on the part of the other nerves of special sense are likewise not uncommon. In the patient whose case (No. 48) has been described on p. 142, the first symptoms of illness were loss of taste and double vision, which proved temporary in character. In the following case there was anosmia and deafness:—

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[1] "Centralblatt für Nervenheilkunde." June 1, 1884.

Case 82.—A clerk, aged thirty-four, married and father of one child, was sent to me by Dr. Hill, of Abbey Road, in March, 1884. Sixteen years ago he had had a severe attack of rheumatic fever, which left great weakness and a cardiac affection. He was obliged to be away from business altogether for twelve months. Some time afterwards he began to suffer from dyspepsia and severe attacks of vomiting; he sometimes vomited incessantly for a whole day, but eventually got better. In December, 1882, however, he had a return of the vomiting, and suffered from hæmorrhage from the mouth, passed blood with his urine, and had purpura stains on his skin. At present he complains of impaired memory, confused feeling in his head, and startings in his sleep. About three months ago he found that he had lost his sexual power. The bowels at the same time became confined, and occasionally do not act for four or five days; the bladder is sluggish. He has habitually to wait five minutes before he can pass any water; then it comes drop by drop, and the performance takes altogether another ten minutes. He has a feeling of tightness across his chest; has occasionally had slight shooting pains in the legs; has slight numbness in the feet, staggers on closing his eyes; cannot stand well on one leg, or go downstairs, and is very unsteady in walking. He has, however, lately walked as much as eight to ten miles on a stretch. The knee-jerk is greatly exaggerated in the right, and normal in the left leg. There is no ankle-clonus, and the excitability of the quadriceps is not increased. His hands are shaky, and he has difficulty in writing. There is some *degree of ptosis in the left eye; he is completely deaf in the left ear,* where he hears neither tuning-fork nor watch, *and has lost the smell in the left nostril;* while there no ophthalmoscopic or other sign of optic atrophy.

This patient recovered almost completely, under the influence of iodide of potassium and nitrate of silver, in about four months; and it is therefore possible that the case may have been not one of multiple but of "pseudo-sclerosis." (*Vide* Chapter XVI.)

The *deep reflexes* may be increased, lost or normal; and this depends entirely upon the localisation of the morbid process. Where patches occur in the lumbar portion of Burdach's columns, the reflexes are lost; while if this be spared, and the lateral columns are chiefly affected, exaggeration of the patellar and other deep reflexes must be the result. The latter is by far the most common occurrence.

The *bladder* suffers in many cases of multiple sclerosis. In Case 81, p. 373, there had been incontinence; in Case 80, p. 372, paresis with ammoniacal decomposition of the urine; in Case 82 extreme sluggishness of the expulsive power of the bladder; in Case 50, p. 144, great irritability of the viscus, so that the patient felt constant desire to pass his water, and wetted the bed in his sleep. In other cases, however, where localisation is different, no symptoms on the part of the bladder may be observed for years.

The *bowels* are frequently confined, and the sexual power is often lost (Case 80, p. 372). In a case which was under my care some years ago, difficulties of *menstruation* were one of the first symptoms of the illness:—

Case 83.—A single lady, aged twenty-eight, consulted me in January, 1877. Her illness commenced about three years ago, with double vision, which, however, went off again after a few months. In that year the period became irregular, apparently without any cause, as she missed it twice. The year after that she only had it twice altogether, viz., in May and November; and now it is entirely gone. Two years ago her sight became much weaker; and at present there is some degree of optic atrophy, temporal limitation of the visual field, dimness of sight, and dyschromatopsia. There is numbness in the little and ring finger of the left hand, but no actual loss of power. She complains of a feeling of tightness round the chest. Her walk is uncertain; she staggers very much, cannot readily put her foot on a chair, or stand on one leg without support; nor can she separate the feet when they are close together. She sways to and fro on closing her eyes. The bladder is atonic; she habitually goes twelve hours without passing water, and then only does it because she thinks it right to do so. When she wants to pass it, she has to wait about ten minutes, and strain all the time as hard as possible, and is then about ten minutes longer in passing it, drop by drop. The bowels are likewise obstinate, and never act without medicines or injections. *All the deep reflexes are greatly exaggerated.*

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Such are the symptoms which may be observed in the first stage of multiple sclerosis; and this stage may last from two to six years.

The second period of the disease is characterised by aggravation of the symptoms of the first, and the appearance of spastic paralysis. The patient, who has until then been able to walk about, although with difficulty, is now reduced to the condition of a confirmed invalid. The legs are held in a state of adduction, and the feet show the condition of varo-equinus. The deep reflexes are considerably exaggerated. The upper extremities are generally less severely affected than the lower ones. This stage may likewise last a considerable time, viz., from two to ten years.

In the third period of the disease, all the functions of organic life show signs of gradual failure. There is more or less complete anorexia, diarrhœa, and general emaciation. The mind becomes more dull and confused, and the speech more unintelligible, until the patient is only able to grunt. Apoplectiform and epileptiform seizures, which we have seen to occur occasionally in the first stage, are now very common. Death may ensue, with symptoms of deep coma, and the appearance of an acute bedsore on the sacrum; or the patient may once more rally, and succumb to a subsequent attack. In other cases there is complete marasmus; paralysis of the sphincters; gangrene of the bladder; bedsores in various parts, and blood-poisoning; or the patient is carried off by intercurrent diseases, such as pneumonia, phthisis, and dysentery.

* * * * *

This *diagnosis* of multiple sclerosis is, like those of many other spinal diseases, sometimes very easy and at other times exceedingly difficult. We have already discussed the peculiar features by which it may be distinguished from *shaking palsy* and *chorea* (p. 368). From the description of the cases which have been given in this chapter, it will be seen that multiple sclerosis occasionally resembles *tabes* in many particulars. There may be initial apoplectiform seizures; failure of mental power; temporary or permanent affections of the sensorial nerves; various forms of anæsthesia and paræsthesia; a gait which, if not absolutely identical with the ataxic, may at least closely resemble it; and troubles in the sphere of the bladder, rectum, and sexual organs, as well as gastric crises. In a number of cases, therefore, we shall be obliged to rely altogether on Westphal's and Argyll-Robertson's symptoms, both of which are habitually absent in multiple sclerosis. Lightning-pains are likewise exceedingly rare, and, if present, not of the violent aud persistent character which they habitually assume in tabes.

The diagnosis between *Friedreich's disease* and multiple sclerosis may occasionally present almost insuperable difficulties: where the latter occurs in children or persons about the age of puberty, and affects the posterior columns with preference, such a distinction may indeed be impossible. In most cases, however, the fact that in Friedreich's disease the principal symptom is ataxy, and in multiple sclerosis tremor, as well as the opposite state of the deep reflexes in the two maladies, will be sufficient for the diagnosis.

Spastic spinal paralysis resembles multiple sclerosis more especially in the second stage, when there is paresis, rigidity, and increase of the deep reflexes. The absence of the peculiar tremor and of head-symptoms will generally speak in favour of spastic paralysis.

A symptom which I consider of great diagnostic importance is that in multiple sclerosis the deep reflexes are often quite different on both sides. In tabes the anatomical lesion is invariably bilateral; and although one side of the cord is often more severely damaged than the other, yet even the less affected side has been so much altered as to cause the knee-jerk to disappear. There is therefore equal loss of patellar reflex in both sides. In spastic spinal paralysis the degree of exaggeration of the deep reflexes varies occasionally, but not habitually, while in multiple sclerosis, where the lesions are the reverse of uniform, decided differences in degree are the rule in the two sides.

Pseudo-sclerosis (p. 67) may assume many symptoms of multiple sclerosis, and the diagnosis between the two conditions would appear to be occasionally almost, if not quite, impossible.

The *prognosis* of multiple sclerosis is bad when the malady has become fully developed. In the earlier stages of it, however, it is better. The patient whose case (No. 82) is described on p. 375 recovered, under the influence of nitrate of silver and iodide of potassium, from all symptoms excepting the unilateral anosmia and deafness, which remained stationary, but did not trouble him much.

The *treatment* of multiple sclerosis is still in its infancy;

and it has no doubt been hitherto unsuccessful because remedies of which good may be expected are generally employed only after so much damage has been done that perfect recovery is out of the question. Iodide of potassium, arsenic, and nitrate of silver should be successively administered, while a careful use of electricity is advisable for the purpose of strengthening the central nervous system against further inroads of the disease.

CHAPTER XVI.

PSEUDO-SCLEROSIS.

PSEUDO-SCLEROSIS is a term proposed by Westphal for those probably not very uncommon cases where serious symptoms of nervous disturbance occur, resembling in all respects certain forms of sclerosis with which we are familiar, and where yet after death no palpable lesions are discovered in any portion of the nervous system. To the cases which I have already given (p. 67) the following may be added:—

A midwife, aged thirty-nine, who had for several years suffered from headache, vertigo, tremor, and paresis in the lower extremities, was, on admission into the General Hospital at Vienna, found to have drawling speech, and to answer questions in an unconnected manner. Convulsions were occasionally seen in the muscles of the body and the lower extremities; there was ataxy in the left hand, and paresis of the legs. On attempting to raise the legs in bed or to bend the knee, muscular spasms supervened. The patient died a few days afterwards, and nothing abnormal was discovered in any portion of the nervous system.

The following case, which occurred in my practice a short time ago, was probably one of pseudo-sclerosis:—

Case 84.—A married woman, aged thirty-eight, and mother of five children, came under my care at the hospital in April, 1883. She attributed her illness to having been sitting in her shop, while alterations were going on, in very cold weather, and having felt constant chills to hei back during that time. Shortly afterwards she began to have shooting-pains, like electric shocks, running through the shoulders, and more particularly through the right arm. She also complained of numbness in the third and little finger of both hands; and, on testing the parts with a pin and the æsthesiometer, a considerable degree of anæsthesia and analgesia was ascertained. She had difficulty in writing, and she could hardly button her dress on account of the awkward feeling in her fingers. She had great difficulty in walking, could not walk longer than ten minutes, and felt numbness in the lower extremities. Romberg's symptom was well marked. The knee-jerk was greatly exaggerated in both legs. Menstruation was regular; the bladder and rectum appeared in their normal condition; and there was no affection of the brain and the cranial nerves. I put the patient on iodide of potassium, under which she began to improve almost immediately; and when I saw her last, about six months afterwards, she was in all respects in perfect health.

I will only add that this chapter, like that on sclerosis of Goll's columns, has yet to be written.

Ballet and Minor[1] have lately used the term "fausse sclérose" for combined disease of the posterior and lateral columns; and as this would be sure to lead to confusion, it is to be hoped that their example will not be followed.

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[1] "Archives de Neurologie," vol. vii., p. 44. Paris, 1884.

CHAPTER XVII.

COMBINED POSTERO-LATERAL SCLEROSIS.

THIS is at present one of the least studied chapters of spinal pathology, but signs are not wanting that our knowledge of it will soon be much more extended than it is now.

There are at present only about a dozen cases on record which point to the existence of this disease as a separate entity. These cases have been described, in chronological order, by Kahler and Pick,[1] Prévost,[2] Westphal,[3] Rabesiu,[4] Hamilton,[5] Raymond,[6] Damaschino,[7] and Ballet and Minor.[8]

In analysing these cases, which present considerable varieties of symptoms as well as of anatomical lesions, it appears that we have in all of them to do with a simultaneous affection of the posterior and lateral columns, or portions of them, without any simultaneous lesion of the central grey matter. There is, therefore, no simple combination of tabes with amyotrophic lateral sclerosis, but something essentially different. In fact, there seems to be every possible combination which one might be led to expect, as far as localisation is concerned, while, on the other hand, the nature of the morbid process also differs considerably.

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[1] "Archiv für Psychiatrie," vol. viii., p. 251, and vol. x., p. 179. 1877.

[2] "Archives de Physiologie normale et pathologique," p. 764. Paris, 1877.

[3] "Archiv für Psychiatrie," vol. ix., pp. 413, 691. 1878.

[4] Virchow's "Archiv," vol. lxxvi., p. 74. Berlin, 1879.

[5] "Medical Record," vol. xv., p. 481. New York, 1879.

[6] "Archives de Physiologie normale," etc., No. 7. Paris, 1882.

[7] "Comptes Rendus," etc. Paris, 1882.

[8] "Archives de Neurologie," vol. vii., p. 44. Paris, 1884.

1. Perhaps the simplest form of postero-lateral sclerosis is one where we find the ordinary lesions of tabes combined with wasting of the direct cerebellar columns. This constitutes a decided system-disease, inasmuch as it spares all the other parts of the lateral columns, with the only exception of the direct cerebellar strands (p. 25).

2. Another form consists of sclerosis of Burdach's columns, which after a time becomes complicated with inflammation of the pia mater. This is a common occurrence in tabes (pp. 14 and 42) ; but while in general the leptomeningitis appears to have no further influence, the inflammation in these cases spreads from the pia mater to the lateral columns in a more or less diffuse manner (cases of Prévost, Raymond, and Westphal).

3. A third form, which is again a true system-disease, consists of simultaneous sclerosis of the entire posterior and lateral columns, together with Türck's direct pyramidal column, which was found affected in the cervical and dorsal region of the right side of the cord (case of Kahler and Pick). In this case, however, the subject of which was an ill-developed girl of twenty years of age, it is possible that we may have had rather to do with incomplete development than with actual disease of the spinal centre (p. 13).

4. A further form of postero-lateral sclerosis is one where Burdach's columns appear affected chiefly in their dorsal, and much less in their lumbar portion, while Goll's columns are found diseased in the cervical region. The lateral columns, in their turn, are not systematically sclerosed, either in the direct cerebellar or the crossed pyramidal strands ; but there is irregular, diffuse, and unsystematic destruction of them, resembling more multiple

sclerosis than system-disease. The wasting is seen to proceed from the pia mater towards the centre of the cord, traversing the direct cerebellar column, and encroaching upon the crossed pyramidal strand. Wasting of the optic nerves has accompanied this singular lesion (cases of Rabesiu and Ballet and Minor).

5. Finally, it appears that diffuse myelitis, limited to a certain area of the cord, may lead to systematic disease of Goll's columns above the seat of the lesion, and of the pyramidal strands below it. To this may be added sclerosis of the direct cerebellar columns. (Cases of Gulliard, quoted by Ballet and Minor, of Pierret and Westphal.) It will be seen that these cases differ completely from multiple sclerosis, which never causes any secondary degenerations, either above or below the seat of the lesion.

The symptoms of all these different forms of sclerosis appear to be very similar, and constitute, as it were, the response of those portions of the posterior and lateral columns, which may appear to be affected, to the morbid process. The nature of the lesion is here of much less importance than its localisation. On the one hand, we know the principal signs of posterior sclerosis to consist of affections of sensibility, such as pain, hyperæsthesia, anæsthesia, analgesia, paræsthesia, ataxy, and loss of deep reflexes; while, on the other hand, the chief symptoms of lateral sclerosis have been seen to be muscular rigidity and paresis with exaggerated deep reflexes. Now it is found that, similarly to what we have seen to occur in multiple sclerosis, that lesion which is the most severe and extensive will impart to the case its characteristic aspect, whether posterior or lateral. Thus, where the posterior columns are destroyed in the lumbar portion of the cord, there must be loss of the knee-jerk, with flabby condition of muscles, although there may be a simultaneous lesion of the lateral columns; and, on the contrary, where these and other portions of Burdach's columns are spared, the signs of spastic paralysis will be present. While, therefore, a singular mixture of symptoms may be observed in certain cases, a diagnosis of the principal localisation of the disease will generally be possible as soon as it has become fully established.

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